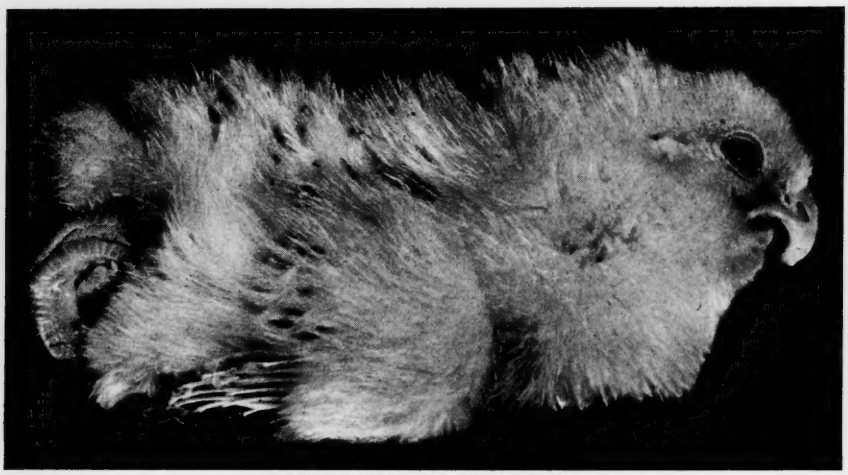
The Journal of HEREDITY



CHICKIE WANT A CRACKER?
(See page 275)

The Sex Ratio in Cattle

A Wavy-Leaf Gene in the Peach

Albinism in the Virginia Deer

Hair-Form of Whites and Indians

Aberrant Ratios in Mouse Crosses

The Need for "Gene Conservatories"

Super-Toenails and Skin Cysts in One Family

VOLUME 33 AUGUST, 1942 NUMBER 8

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President Vice-President, SEWALL WRIGHT Secretary, C. E. LEIGHTY
Treasurer, J. H. KEMPTON
Legal Advisor, STANTON C. PEELLE

EDITORIAL BOARD

Managing Editor, ROBERT C. COOK

M. DEMEREC GEORGE M. DARROW
H. J. MULLER R. R. GRAVES PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

W. E. CASTLE, Emeritus Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.
LEON J. COLE, Professor of Genetics, University of Wisconsin.
O. F. COOK, Bionomist in Charge of Breeding Investigations, U. S. Department of Agriculture.
DAVID FAIRCHILD, Agricultural Explorer, U. S. Department of Agriculture.
HARRY V. HARLAN, Senior Agronomist in Charge of Barley Investigation, U. S. Department of Agriculture.
SAMUEL J. HOLMES, Professor of Zoology, University of California, Berkeley, California.
MORLEY A. JULL, Department of Poultry Husbandry, University of Maryland, College Park, Md.
J. H. KEMPTON, Bureau of Plant Industry, U. S. Department of Agriculture.
W. V. LAMBERT, Associate-Director, Purdue University Agricultural Experiment Station, Lafayette, Indiana.
C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.
MARCUS M. RHOADS, Department of Botany, Columbia University, New York City.
SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.
E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.
E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.
A. F. BLAKESLEE, Plant Geneticist, Carnegie Institution, Cold Spring Harbor, New York.
W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.
C. B. DAVENPORT, Carnegie Institution of Washington, Cold Spring Harbor, L. I., New York.
RUSSELL W. DUCK, Livestock Editor, *The Rural New Yorker*, Syracuse, N. Y.
R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.
C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.
C. M. GOETHE, Sacramento, California.
HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.
H. S. JENNINGS, Professor of Zoology, Johns Hopkins University, Baltimore, Maryland.
D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.
M. A. JULL, Senior Poultry Husbandman, U. S. Department of Agriculture, Washington, D. C.
F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.
C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.
JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.
H. H. NEWMAN, Professor of Zoology, University of Chicago, Chicago, Ill.
T. S. PALMER, Expert in Game Conservation, U. S Dept. of Agriculture, Washington, D. C.
JOHN H. PARKER, Professor of Agronomy, Kansas State College, Manhattan, Kansas.
JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.
A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.
GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.
EDMUND W. SINNOTT, Professor of Botany, Barnard College, Columbia University, New York City. (Representative of A. G. A. on Council of Amer. Assoc. for the Advancement of Science.)
W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.
A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.
W. T. SWINGLE, Plant Physiologist in Charge of Breeding Investigations, U. S. Department of Agriculture, Washington, D. C.
W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.
H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.
H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.
E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.
P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and 1108 Sixteenth St., N. W., Washington, D. C. Address all communications regarding editorial matters to Room 205, 1108 Sixteenth St., N. W., Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1108 Sixteenth Street, N. W., Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918.

Contents copyrighted 1942 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$3.50 a Year. 35c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXXIII **AUGUST, 1942** **No. 8**

CONTENTS

HEREDITARY CHONDRODYSTROPHY IN THE FOWL
By W. F. Lamoreux 275

HYPERKERATOSIS OF THE LARGE TOENAILS AND SEBACEOUS CYSTS
By J. Stauffer and I. Simmons 285

ABERRENT RATIOS IN PEROMYSCUS
By R. R. Huestis and Victor Piestrak 289

THE NEED FOR "GENE CONSERVATORIES"
By Hans Gruneberg 291

THE INHERITANCE OF WAVY LEAF CHARACTER IN THE PEACH
By D. H. Scott and F. P. Cullinan 293

ON THE SEX RATIO IN CATTLE
By John W. Gowen 299

THE SHAPE AND SIZE OF HUMAN HEAD HAIR
By Henri C. Seibert and Morris Steggerda 302

Notes and Reviews

ALBINISM IN THE VIRGINIA DEER 274

GENETICS AND THE ORIGIN OF SPECIES (Rev. of DOBZHANSKY'S SECOND EDITION)
By Sewall Wright 283

EFFECTS OF THE WAR ON BRITISH FERTILITY 288

A BOOK OF THE PIGEON (Rev. of LEVI: The Pigeon)
By Leon J. Cole 296

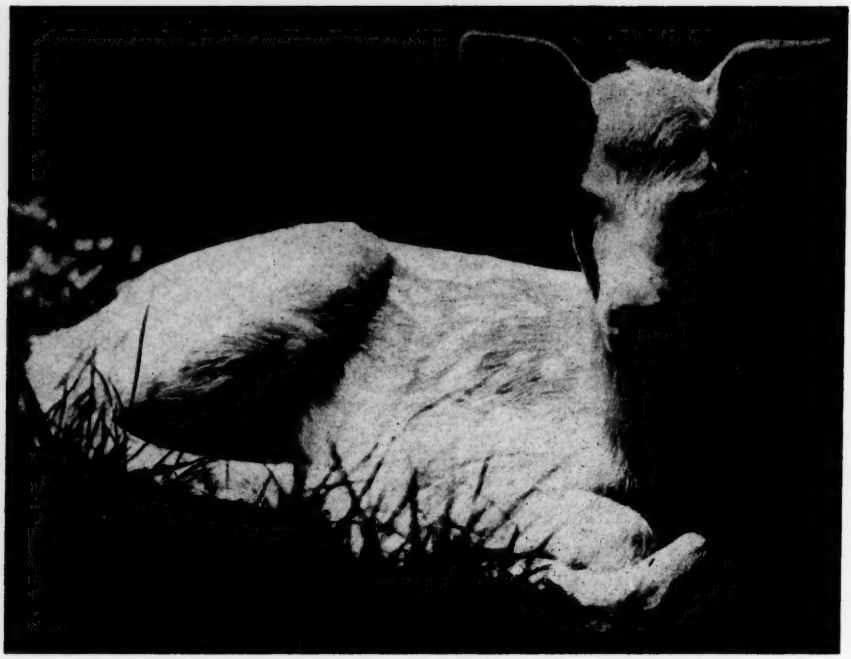
STUDENT SUBSCRIPTIONS

The **Journal** has obvious advantages as a supplementary text in genetics classes. Since genetics deals mainly with form and pattern differences in organisms the **Journal** offers an unexcelled introduction to current developments, which have an added interest and appeal on account of the large number of illustrations published.

Some years ago we tried the experiment of offering the **Journal** at a special student group subscription rate for classroom use. The plan was tried the first year by several of our members, and some of them are still using it every year, a convincing testimony to its value as a supplementary text.

STUDENT SUBSCRIPTIONS covering the academic year of nine months in groups of ten or more, may be ordered by members at one dollar ($1.00) each; four months for 50 cents. These special rates to students will be accepted only when applied for by members of the Association, in whose classes the **Journals** are to be used. The student subscriptions will be sent in bulk to the member in whose name they are ordered. Orders should be sent to the Washington office of the Association.

$3.50 a Year **35c the Copy**



AN ALBINO FAWN
Figure 1

An albino buck captured in 1935, when mated to normally pigmented does produced colored fawns. Crossing the albino to his daughters produced a number of albino fawns. As is so often the case with albino individuals in a wild species, few of these have survived to adulthood. Under normal conditions among mammals it appears that albinism is essentially a lethal characteristic.

ALBINISM IN THE VIRGINIA DEER

A NEWSPAPER photograph of an albino fawn from Wisconsin brought to light an interesting experiment on the inheritance of albinism in this species conducted by Mr. H. B. Kellogg, Jr., manager of the Wisconsin State Game Farm at Poynette, Wisconsin. The evidence as far as it goes is consistent with the view that albinism in this species, as elsewhere among mammals, is inherited recessively. We quote from Mr. Kellogg's account of the albino matings at the Poynette Farm:

In the spring of 1935, a buck was picked up in the vicinity of Bayfield, Wisconsin, which proved to be a true albino of the Virginia whitetail deer. He was brought to our farm at Poynette and has been here ever since.

After some little trouble at first, he grew normally and today is a fairly good sized specimen of deer. As we are the clearing house for the Conservation Department in the matter of confiscated wild animals, we usually have deer on our farm. We immediately became interested in albinism and decided to carry on an experiment with him and his offspring.

We left him in a pen with several doe deer and in 1936 he mated and produced fawns which were normal in size and color. In 1937 this also proved to be the case. In 1938 we mated this white buck back to several of his own offspring. These were also normal in size and color. In 1939, however, a doe from his own offspring produced a white fawn which was a true albino and one which we were un-

(*Continued on page* 284)

HEREDITARY CHONDRODYSTROPHY IN THE FOWL

W. F. LAMOREUX
Department of Poultry Husbandry, Cornell University, Ithaca, N. Y.

CHONDRODYSTROPHIC chick embryos were first reported in 1923 by Dunn.[1] His description of the "parrot" embryos indicates that the abnormality appeared in an extreme form. Since it was found only among the progeny of one male and a female (in the proportion of five "parrots" to nineteen normals) Dunn suspected that the character was inherited as a simple recessive. Extensive studies were then made to determine accurately the genetic nature of chondrodystrophy, but by 1927 he reported[2] that the accumulated evidence "made it appear doubtful whether the condition is influenced in any way by inheritance."

The following year Hutt and Greenwood[4] reported a study of 124 chondrodystrophic embryos produced by 22 hens in Scotland. One of these hens produced 28 chondrodystrophic embryos among 61 classified. Since the probability is very small that such a high incidence of a non-genetic character could occur by chance, the authors believed the causal agency to be an "hereditary physiological abnormality in the dam" which, under unfavorable environmental conditions, results in the production of chondrodystrophic embryos.

A study of chondrodystrophy was later made in Canada by Munro.[8] He also was unable to explain its occurrence on a unifactorial genetic basis, but he did present additional evidence that the incidence of chondrodystrophy is higher during February and March than during April, May, and June. These results support the original suggestion by Hutt and Greenwood[4] that the length of the daily period of sunlight may influence the incidence of chondrodystrophy. Since that time, chondrodystrophic embryos have been observed at various times in other widely separated places, but no evidence has been reported to indicate a definite genetic basis for this defect.

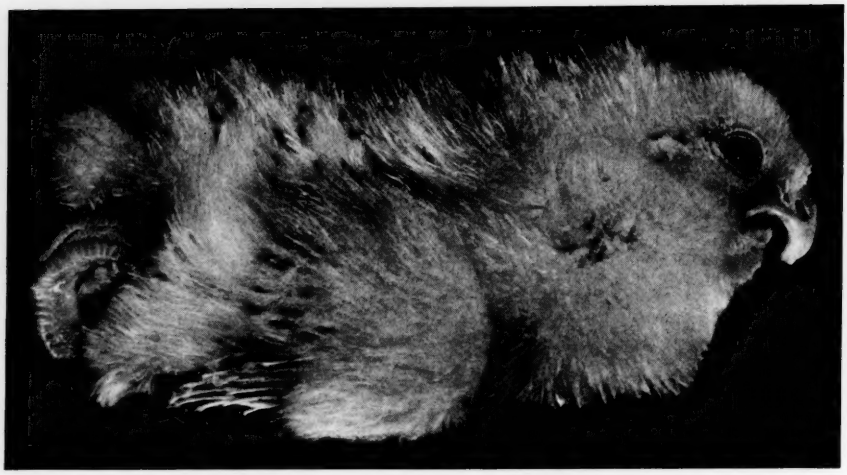
During the routine examination of the embryos which failed to hatch at Cornell in 1936 and 1937, several extremely chondrodystrophic chicks were found among the progeny of two closely related hens in an inbred strain of White Leghorns. Other related hens were then mated with a sire of chondrodystrophic chicks and in this way seven additional hens were found which produced affected embryos. These mature birds and their surviving progeny were then used in a study of the inheritance of chondrodystrophy.

Description of Chondrodystrophy

Two distinct types of chondrodystrophic embryos have been observed in this laboratory. The first, and probably the one most commonly observed elsewhere, might be called a "sporadic" type. Affected embryos commonly die before the nineteenth day of incubation and even when found alive they appear to be somewhat retarded in their development when compared with normal embryos the same age.[4]

The second type, and the one with which this study is concerned, shows all the typical morphological characteristics of the sporadic type as described by previous authors, but they show no apparent retardation of growth during incubation. Such embryos are unable to hatch without assistance because of the deformities of the beak and legs, but many are found alive on the twenty-first and twenty-second days of incubation.

Morphology. An extremely chondrodystrophic day-old chick which was given assistance in hatching is illustrated here in Figure 2. The short lower and the curved upper mandibles, the shortened and bent tibiae (not visible) and



EXTREME CHONDRODRYSTROPHY

Figure 2

An extremely chondrodystrophic day-old chick which was given assistance in hatching. The short lower and the curved upper mandibles, the shortened and bent tibia (not visible) and tarso-metatarsus are typical of this abnormality which is inherited as an autosomal recessive. This chick was quite unable to stand.

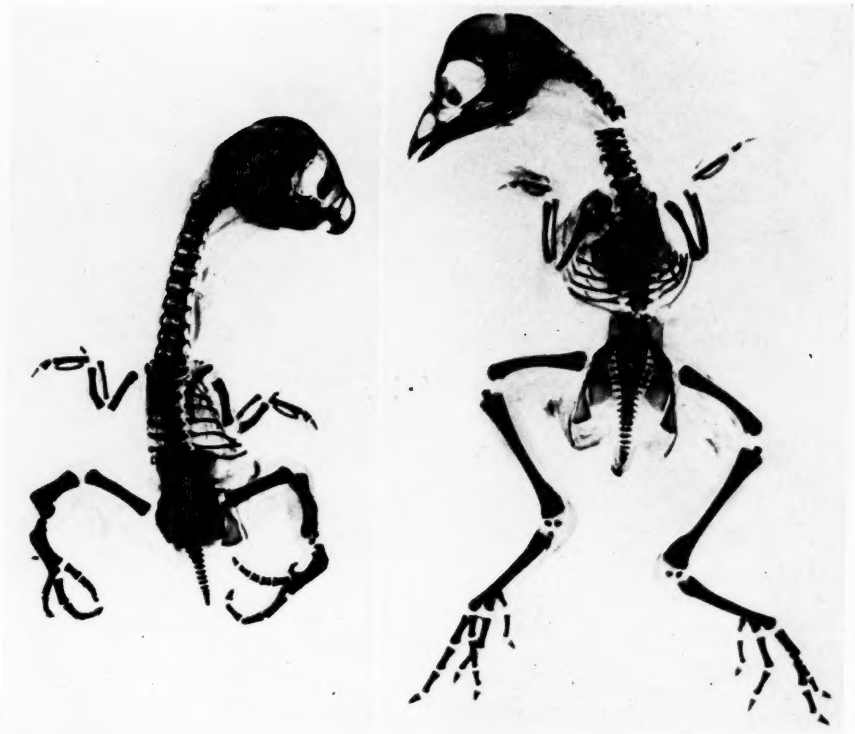
tarso-metatarsi are typical of this abnormality. This chick was quite unable to stand.

Better to illustrate the skeletal modifications associated with the character, a chondrodystrophic and a normal embryo were obtained on the twenty-first day of incubation, cleared and stained with alizarin. The two embryos were photographed at the same magnification and are shown in Figure 3 for comparison. A further comparison was made by obtaining measurements of one femur, tibia, and tarso-metatarsus from each of five male and five female chondrodystrophic embryos 21 days of age and from the same number of normal embryos. The average lengths of these bones are given in Table I to show the degree to which they are shortened. A slight curvature and a similar but less marked shortening of the bones in the wing can also be noted in Figure 3. The relative size of the head and cervical vertebrae appears unchanged, but the marked shortening and curvature of the beak are apparent.

Probably because of the reduced length of the appendages, many of the chondrodystrophic embryos appear to carry a disproportionate amount of yolk in the body cavity. A study of the embryos considered in Table I showed that 23.5 per cent of the body weight of chondrodystrophic chicks was yolk, as compared with 20.4 per cent of the normals. However, an analysis of variance (percentages transformed to equivalent angles) showed that with variation as great as was found in these groups a difference no greater than the one obtained would not be significant even though an infinite number of chicks were considered.

The histology of the abnormality has not been studied at Cornell. Dr. Walter Landauer[5] described the morphology and histology of the sporadic type of chondrodystrophy in 1927 and he has kindly given the following description of chondrodystrophic embryos obtained in this study:

A preliminary histological examination of some of the long bones (femur, tibia) of em-



SKELETONS OF CHONDRODYSTROPHIC AND NORMAL EMBRYOS
Figure 3

Left. An extremely chondrodystrophic embryo after 21 days of incubation. It has been cleared and stained with alizarin. Note particularly the short curved beak and the extremely shortened and bent tibiae and tarso-metatarsi. Right. A normal embryo the same age.

bryos of the "extreme" type indicates that the structural abnormalities in the skeleton of these embryos are similar in nature to those previously described for sporadic chondrodystrophy of chicken embryos. In both kinds of material the most striking changes consist in great irregularity of cell arrangement and of cell formation in the zone of columnar cartilage of the epiphyses. Another conspicuous feature which the two groups of embryos have in common is a sharp bend in the tibia shaft. The degree of shortening of the long bones seems to parallel closely the extent of histological abnormality of the epiphyseal cartilage. On the whole, the histological features of your material warrant, I believe, the diagnosis "chondrodystrophy."

Variation. The expression of chondrodystrophy ranges from the type illustrated in Figure 2 to embryos barely distinguishable from normal. A few of the latter are able to hatch without assistance, but in general their viability has been low. One was reared to maturity and used for a short time in a breeding pen. Another which exhibited a slight curvature of the upper mandible and the tibiae at hatching is illustrated in Figure 4 at the age of eight weeks when the abnormality of the beak was still apparent.

Classification. To facilitate study of the chondrodystrophic embryos, they have been classified as either "extreme" (Figures 2 and 3) or "modified" (Figure 4), depending upon the degree in which the character is expressed. Em-

bryos which died before the eleventh day of incubation were not classified.

Genetic Basis for Chondrodystrophy

In 1936 the hen which was first found to produce the extreme type of chondrodystrophic embryos produced four extreme, two modified, and 32 normal embryos or chicks. The following year eight related hens produced 39 extreme, 17 modified, and 197 normal chicks. This incidence of more than 21 per cent chondrodystrophic chicks suggested immediately that the character behaved as a genetic recessive.

Carrier ♀ ♀ × Carrier ♂ ♂. Altogether, five generations of birds have been studied in an attempt to determine the genetic basis for this type of chondrodystrophy. The proportion of affected embryos produced by matings between males and females known to have produced extremely chondrodystrophic embryos is shown in Table II. When the five years' data are summarized, the proportion of affected embryos (22.5 per cent) is reasonably close to the 25 per cent expected of a simple autosomal recessive gene. It is proposed to designate this gene with the symbol, *ch*, which, so far as the author can ascertain, has not been used for any other genetic character in the fowl.

Carriers among the progeny. One would expect from such matings that two of every three normal daughters would be heterozygous with respect to chondrodystrophy. Actually, among 61 such daughters there were 15 non-carriers, 13 with modified, and 33 with extremely chondrodystrophic embryos. Most of those hens which had modified chondrodystrophic progeny, but none extreme, undoubtedly were carriers in whose offspring modifying factors, genetic or otherwise, had limited expression of the character. Actually, the 46 hens that produced chondrodystrophic embryos can all be considered true carriers and the ratio of 15:46 does not deviate significantly (Chi Square value for $P = > .05$) from the 1:2 ratio expected among the progeny of heterozygous parents.

Different ♂ ♂ mated to the same hens. If the character is a simple recessive, one would also expect that different carrier males would give comparable results when mated with the same group of carrier females. This comparison was possible between two males which when mated to the same 11 hens gave the following almost identical proportions of chondrodystrophic embryos:

	Classified embryos (number)	Chondrodystrophic embryos (per cent)
♂ E5806	179	24.0
♂ G5148	113	24.8

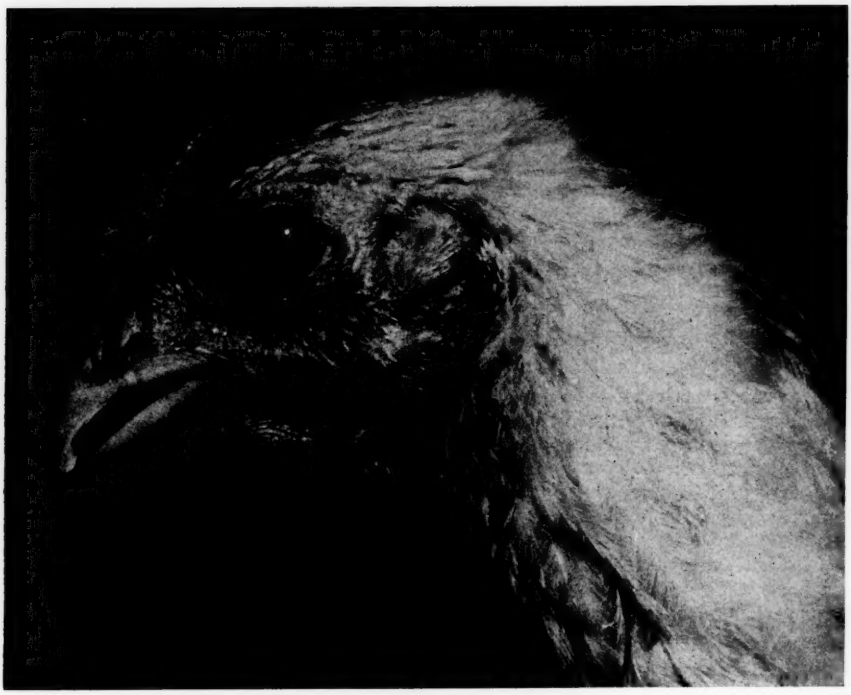
A similar comparison has been made between carrier and non-carrier males of the same strain mated, at different times, to the same five carrier females.

	Classified embryos (number)	Chondrodystrophic embryos (per cent)
Carrier ♂ ♂	94	30.7
Non-carrier ♂ ♂	94	2.1

Only two chondrodystrophic embryos were found among the progeny of the non-carrier males and neither of these were of the extreme type. This difference of 28.6 per cent between the progenies of the two groups of males is close to the 25 per cent expected if a recessive gene (*ch*) was carried by males in the first group but not by those in the second.

Modified phenotype. It is not known what may influence the expression of chondrodystrophy, but previous studies indicate[2,4,8] that important non-genetic factors are involved.

One might suspect from the variation found in the expression of the character that occasionally under very unfavorable conditions a heterozygote may appear as a modified individual, whereas under very favorable conditions a homozygote may also appear as a modified chondrodystrophic chick, hatch and survive. Some evidence that the latter may occur was obtained from male H5077. This bird had been classified as a modified chondrodystrophic chick, was reared to maturity, and mated with five known carrier hens. Unfortunately, the male died before a large number of progeny



MODIFIED CHONDRODYSTROPHY

Figure 4

The expression of chondrodystrophy ranges from the type illustrated in Figure 2 to embryos scarcely distinguishable from normal. A few of the latter are able to hatch without assistance, but in general their viability has been low. This picture illustrates such a chick at eight weeks of age when the abnormal curvature of the upper mandible was still apparent. Another such chick was reared to maturity and used for a short time in a breeding pen.

could be obtained, but among 69 classified embryos from these matings 35 were normal and 33 (24 extreme : 9 modified) were chondrodystrophic.

This 1:1 ratio, conforming to that expected when heterozygotes are backcrossed to a homozygote, shows that ♂ H5077 was probably homozygous for the mutation. From the facts that very few of the chondrodystrophic embryos hatched and that the proportion of such embryos was only 2.5 per cent less than expected of a recessive character (Table II), it is probable that the survival of this homozygous (*chch*) male was a rare exception to the rule that homozygosity for this type of chondrodystrophy is lethal, if the parents have produced extremely chondrodystrophic embryos.

However, if dams are used which produce the modified type but no extremely chondrodystrophic chicks, there is a considerable deficiency of chondrodystrophy among the progeny. When the records of the 13 such hens mentioned above are summarized, only 13.8 per cent of their 254 classified embryos were found to be chondrodystrophic. From these hens there were 127 fertile eggs in which the embryos died before the eleventh day of incubation. One or more of three explanations might therefore account for the deficiency of chondrodystrophic embryos from these hens:

(1) The extremely chondrodystrophic embryos may have died during the first ten days of incubation. Comparison of the above three groups of hens shows that the embryonic mortality among their fertile eggs during the first ten days of incubation was as follows:

	Fertile eggs (number)	Mortality (per cent)
I—15 hens, normal progeny	266	14.3
II—33 hens, extremely chondrodystrophic progeny	943	25.9
III—13 hens, modified chondrodystrophic progeny	381	33.3

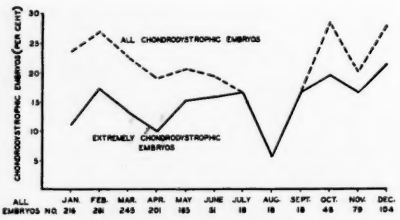
The relatively high embryonic mortality in Group III indicates that at least part of the deficiency in chondrodystrophic embryos from these hens results from the early death of some of the affected embryos, presumably those of the extreme type.

(2) It is also possible that some of the hens in Group III produced no extremely chondrodystrophic chicks because modifying factors inhibited expression of the character to such a degree that no extreme chondrodystrophy developed and some of the genetically chondrodystrophic chicks appeared to be normal.

(3) Presumably the sporadic type of chondrodystrophy would occur in this stock as frequently as elsewhere and when it occurred it would be classified as the modified type. This error would place hens in Group III that should be classified in Group I, thus accounting for part of the deficiency in the number of dams in Group I, as well as for part of the deficiency of chondrodystrophic embryos in Group III.

Seasonal Incidence of Chondrodystrophy

Previous investigators[2, 4, 8] have noted a marked decline in the incidence of chondrodystrophy between January or February and the following summer. This and other irregularities in the manifestation of chondrodystrophy led to the general belief that it was a genetic character expressed only under certain, and



SEASONAL VARIATION

Figure 5

The proportion of the embryos which are chondrodystrophic in eggs laid during different months of the year. Unlike the sporadic type of chondrodystrophy previously known, the incidence of hereditary chondrodystrophy is affected little by the change of seasons.

unknown, unfavorable conditions. A consideration of seasonal variation in this study is important because, if the incidence is significantly higher at certain seasons than can be explained on the genetic basis suggested, it is possible that the ratios obtained by summarizing the data are merely a result of chance combinations of populations having high and low incidence of chondrodystrophy. Regardless of the "goodness of fit," the final ratio obtained in such a way could have no genetic significance.

Since different matings vary in their production of chondrodystrophy, the ideal way to measure the influence of different seasons upon the incidence of the character would be to hatch from the same hens throughout the year. This was not feasible, but data from matings made during 1938 to 1940 have been summarized in Figure 5. There was considerable variation in the incidence of chondrodystrophy from month to month, but no seasonal trend is apparent in the proportion of extremely chondrodystrophic embryos.

There is some indication in Figure 5 that if *any* seasonal influence is exerted it is reflected primarily in the incidence of the modified type of chondrodystrophy. During July, August, and September no modified embryos were obtained. It is true that relatively few chondrodystrophic chicks were found during these months, but the numbers of

chicks involved (18) were too small to show that a real deficiency of affected chicks can be expected during the summer. Furthermore, January, found by others[4] to be a month of high incidence of sporadic chondrodystrophy, ranks third lowest among the twelve months with respect to incidence of extreme chondrodystrophy. July, September, and June, on the other hand, ranked fourth to sixth from the highest.

While environmental conditions undoubtedly play an important role in the manifestation of the original type of sporadic chondrodystrophy studied by Dunn,[2] Hutt and Greenwood,[4] and Munro,[8] it is clear that changes of the seasons do not materially influence the manifestation of the simple recessive chondrodystrophy described in this paper.

Sex of Chondrodystrophic Embryos

Neither Dunn[2] nor Hutt and Greenwood[4] found any significant difference between the sexes with respect to the incidence of sporadic chondrodystrophy. However, Munro[8] found significantly more chondrodystrophic males than females. Since the interpretation of the genetic ratios in Table II might be affected materially if most of the chondrodystrophic embryos were either male or female, it is necessary in this study to consider the sex of affected individuals.

The proportion of males and females among chondrodystrophic and normal embryos was obtained by tabulating those embryos in weekly "sets" of eggs for which the sex was known for all chondrodystrophic embryos and all or most of the normal embryos more than ten days of age.

This method of sampling gives a relatively high proportion of chondrodystrophic embryos because in the incubation records of most hens there are weeks when no affected embryos are found. Thus, a selection of only those weekly periods in which chondrodystrophic embryos occurred must necessarily result in a deficiency of normals. So far as is known, this in no way invalidates the information obtained concerning sex ratios.

In the group of 147 classified embryos of known sex, there was an excess of females among the chondrodystrophic individuals, but this deviation from the sex ratio of the whole group was shown by the Chi Square test to be insignificant (value for $P = > 0.05$). Since there was no known reason for thinking that the sex ratio for the classified embryos not "sexed" would differ from that of the general population, additional weekly "sets" of eggs (excluding 29 classified embryos of unknown sex) were combined with those considered and tabulated with respect to sex and chondrodystrophy as follows:

	Females	Males
Chondrodystrophic embryos	77	73
Normal embryos	87	85

Chi Square, value for $P = > 0.05$

These data give no indication that one sex is more susceptible to hereditary chondrodystrophy than is the other.

Independence of This Chondrodystrophy and Other Types

The variation in expression of the character is so great that one might hesitate to credit a single recessive gene with its control. However, few, if any, simple genetic characters can be shown to be free from some degree of variation and a primary genetic factor controlling

Table I. A Comparison of Normal Chicks With Extremely Chondrodystrophic Siblings on the 21st Day of Incubation. Figures are averages for ten birds, half males and half females.

Weight of chick	Wt. of yolk sac / Wt. of body × 100	Femur (length)	Tibia (length)	Tarso-metatarsus (length)
grams	per cent	mm.	mm.	mm.
Normal 36.5	20.4	20	28	20
Chondrodystrophic 38.1	23.5	15	15	13

Table II. The Proportion of Chondrodystrophic Embryos Produced in Matings Between Carrier Males and Females.

Year	Hens	Embryos more than 10 days of age			
		Normal	"Modified chondros"	"Extreme chondros"	All "chondros"
	no.	no.	no.	no.	per cent
1936	1	32	2	4	15.8
1937	8	197	17	39	22.1
1938	15	291	25	66	23.8
1939	9	162	30	35	25.3
1940	33	645	63	115	21.6
Total	66	1327	137	269	
Per cent			8.0	14.5	22.5

bone development obviously has abundant opportunity for genetic, nutritional, or environmental modification. Furthermore, it has been amply shown by Goldschmidt[8] in his discussion of "phenocopies" that variation, *per se*, is no valid reason for rejecting a hypothesis for simple genetic control of a character. Either genetic or non-genetic modification may explain the fact that some of the daughters of carrier parents do not produce extremely chondrodystrophic embryos, but do produce the modified type.

Sporadic chondrodystrophy. One might suspect that the sporadic type of chondrodystrophy differs from the genetic type only because the latter lacks complicated genetic modifiers which made genetic study of the sporadic type fruitless. However, two lines of evidence indicate that this is not true.

During one year, five hens that had each produced two to nine of the sporadic type of chondrodystrophic embryos (February 13 to April 5) were mated with a male known to carry chondrodystrophy. It was thought that if the two types were related genetically this mating should give at least a few extremely chondrodystrophic chicks, despite the fact that it was made during the summer. The record for these five hens in the diallel crosses was as follows:

	Chondrodystrophic progeny (per cent)
143 embryos from non-carrier ♂ ♂	21.0
89 embryos from carrier ♂	6.7

No extremely chondrodystrophic individuals were obtained.

It is clear that, although about half of the embryos from the carrier male must have gotten from their sire a gene for extreme chondrodystrophy, the proportion of them showing any symptoms of chondrodystrophy was much less than among embryos of the previous mating. Presumably the cases of chondrodystrophy found in both matings were of the so-called sporadic type which is apparently quite independent of the simple recessive type reported in this paper.

This conclusion would be unjustified if sporadic chondrodystrophy differs from the genetic type only because it is associated with dominant genetic modifiers which prevent complete expression of the character in such a mating. However, this is very unlikely, since extreme chondrodystrophy is readily recovered from out-cross populations without any apparent change in severity. This study, therefore, includes no evidence that sporadic and hereditary chondrodystrophy are causally related.

Nutritional micromelia. A third type of micromelia, apparently first described in detail by Landauer,[6] and later shown by Lyons and Insko[7] to result from a deficiency of manganese in the incubating egg, has sometimes been confused with chondrodystrophy, and therefore requires consideration in this study. However, since these two characters are quite different, we need merely note the distinction between them given by Landauer in his description of nutritional micromelia. The chondrodystrophic embryos almost always have a striking bend in the shaft of the tibia. In nutritional micromelia, the tibia always appears to be straight. In nutritional micromelia, "the long bones of the wings are reduced in length to almost the same degree as those of the legs, while in chondrodystrophy the wing bones are much less affected than the leg bones. Histologically, these two types of micromelia are quite distinct." There appears, therefore, to be no reason for confusion between the nutritional micromelia resulting from a deficiency of manganese, and either the sporadic type or the hereditary type of chondrodystrophy in the fowl.

Discussion

Origin. When the pedigrees of the birds which produced chondrodystrophic chicks were examined, it was found that they all had a common male ancestor, B6034, in the generation of 1935. This male's dam was at that time still available, but when she was mated with a male that had produced chondrodystrophic embryos only normal embryos were obtained. The sire of male B6034

was one of six males rotated in an experimental pen during 1934 and then marketed. There is therefore no evidence to indicate whether the character arose by mutation in 1934-35 or had been transmitted through many generations as a recessive. Comparable extremely chondrodystrophic chicks have not been found elsewhere in the station flock, although most of the unhatched eggs have been examined since 1935.

It seems likely that the extreme chondrodystrophy described here is not widespread in its occurrence. Since the first description of chondrodystrophy by Dunn[1] in 1923, several people in widely separated locations have studied the incidence of abnormalities among unhatched embryos, and the genetic bases for different lethal characters they found. Since the chondrodystrophy described here is frequently found among embryos alive on the twenty-first day of incubation, it is highly improbable that any observer would miss the character if it occurred.

Control. This ease of identification facilitates elimination of the lethal by means of test matings, between known and possible carriers, such as are employed for the detection of birds heterozygous for other lethal characters.

Summary

An extreme type of chondrodystrophy has been shown to be hereditary. In five generations of White Leghorns the character was inherited as an autosomal recessive, for which the gene-symbol, *ch,* is suggested. Among 1,713 classified embryos or chicks from heterozygous parents, 22.5 per cent were chondrodystrophic. Unlike the sporadic type of chondrodystrophy previously known, the incidence of hereditary chondrodystrophy is affected little by the change of seasons. Furthermore, hens known to produce sporadic chondrodystrophy did not produce a higher proportion of affected embryos when mated with a male known to be heterozygous (*Chch*) for hereditary chondrodystrophy. There is no significant difference between males and females with respect to the incidence of hereditary chondrodystrophy.

Literature Cited

1. Dunn, L. C. *Scientific Agr.* 4:1-7. 1923.
2. ———. *Wilhelm Roux' Archiv für Entwicklungsmechanik der Organisem.* 110:(341)-365. 1927.
3. Goldschmidt, Richard. Physiological Genetics. McGraw-Hill Book Company, Inc., New York. 375 pages.
4. Hutt, F. B., and A. W. Greenwood. *Proc. Roy. Soc. Edinburgh.* 49, Pt. II: (131)-144. 1928-29.
5. Landauer, Walter. *Wilhelm Roux' Archiv für Entwicklungsmechanik der Organisem.* 110: (195)-278. 1927.
6. ———. *Anatomical Record* 64: 267-276. 1936.
7. Lyons, Malcolm, and W. M. Insko, Jr. *Kent Agr. Exp. Sta. Bul.* 371: (61)-75. 1937.
8. Munro, S. S. *Scientific Agr.* 13: (97)-109. 1932.

✦

GENETICS AND THE ORIGIN OF SPECIES

THE first edition of Dobzhansky's "Genetics and the Origin of Species" appeared in 1937. The publication of a second edition* four years later, testifies to the favor with which it has been received. The addition of 82 pages to the original 364, largely devoted to new data, reminds us of the rapid increase of interest in this field, a development in which the appearance of the first edition played no mean role. Not only has much new material been added but there is evidence of careful revision throughout, including segregation of certain scattered portions into a new chapter, "Patterns of Evolution."

There is no other book which combines such an extensive review and an-

*Dobzhansky, Theodosius. Genetics and the Origin of Species. Second Edition. Pp. 446. $3.50. Columbia University Press, New York. 1941.

alysis of the present state of knowledge on the genetics of variability within wild populations, and of the differences among races and species, with an account of the statistical consequences to be expected from known genetic mechanisms in populations subject to selection, cross-breeding and the cumulative effect of accidents of sampling. There is continual effort to integrate the factual material with the mathematical theory. There is, however, no selection or other forcing of facts to fit theoretical patterns. The primary emphasis is on the facts in all their heterogeneity.

In the author's words, "the aim of the present book is to review the genetic information bearing on the problem of organic diversity, and, as far as possible, to correlate it with the pertinent data furnished by taxonomy, ecology, physiology, and other related disciplines; this book is not concerned with the purely morphological aspect of the problem." The book accordingly does not go much beyond the origin of species into the broader aspects of evolution, to which the application of genetic principles necessarily remains a highly speculative matter. It is largely concerned with "micro-evolution." The author makes it clear, however, that he sees no present necessity for invoking an unknown genetic process such as "systemic mutation" by "repatterning of the chromosomes" urged recently by Goldschmidt, to account for the profound morphological differences and inability to interbreed of "good" species and higher categories. Two chapters in Dobzhansky's book, "Isolating Mechanisms" and "Hybrid Sterility," deal with the last named problem. To him, the available data indicate a cumulative process, the primary phase of which is usually mere geographic or ecologic isolation rather than any genetic change that abruptly isolates a new form.

The evidence for some multiplication of species by an abrupt process, polyploidy, especially allopolyploidy is, however, by no means skimped. A chapter is devoted to a careful review of the data.

The last two chapters, "Patterns of Evolution" and "Species as Natural Units," present effectively the synthesis of facts and known genetic mechanisms that seems warranted today.

A bibliography, which has increased from 29 to 46 pages, gives an indication of the amount of material examined in the preparation of the book. The attention devoted to the work of Russian authors will be especially welcomed by those to whom much of this literature is not easily available.

SEWALL WRIGHT
The University of Chicago

✦

Albinism in the Virginia Deer

(*Continued from page 274*)

able to raise due to the fact that the doe did not take care of the fawn properly and we tried to raise it by hand too late to save it. This white fawn was a buck and is mounted and in our display case at our farm. In 1940, this same doe produced another white fawn, a doe this time, and one which we were fortunate enough to raise to several months of age. This fawn was a beautiful specimen of albinism and we were greatly in hopes that we could raise it to maturity. However, one night the white buck killed this doe fawn.

During this same year another white fawn was also born to one of the does that were mated by the white buck and which we sold to a commercial deer farmer in this state. This farm was quite small and the deer farmer, upon finding it, took it into the house and immediately had trouble in getting the fawn to eat. When we were called concerning it, we were also unable to do anything for it.

In 1941, even though our buck mated the same doe that produced the white fawn in 1939, they produced no fawns.

We still have this doe on our farm as well as a few more of his offspring and will again this year attempt to get a white fawn.

As I stated in a prior letter to you, there is another white doe fawn that will be a yearing this year that was picked up in the northern part of our state and which we may have the opportunity to bring in to our farm and mate with "Whitey."

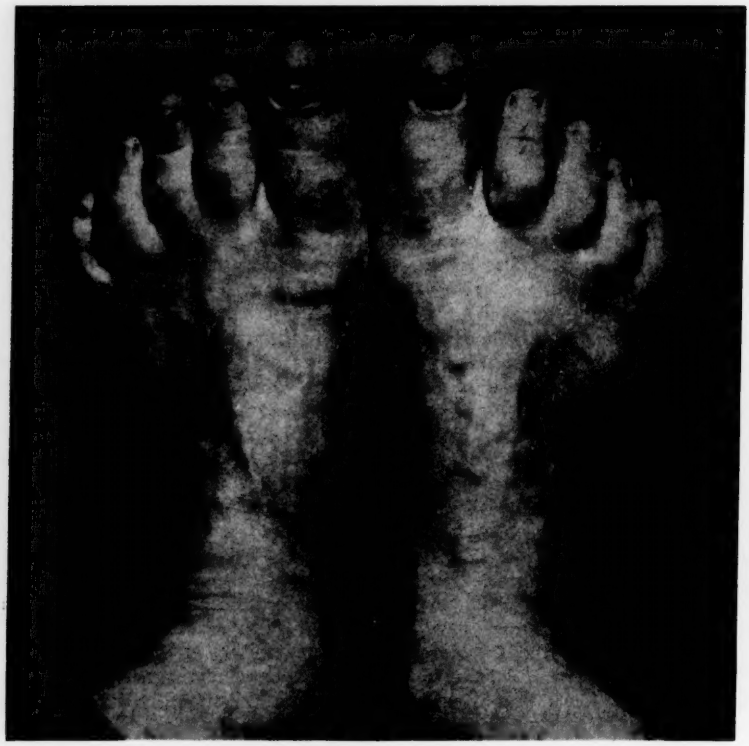
HYPERKERATOSIS OF THE LARGE TOE-NAILS AND SEBACEOUS CYSTS

J. STAUFFER, *Colgate University* AND I. SIMMONS, *Tufts Medical School*

THE accompanying pedigree, showing the inheritance of two interesting human conditions, is noteworthy because the traits in question are first met with separately for two generations in independent branches of the family and are then found associated in a number of persons of the third and fourth generations. Since several of the affected individuals were doctors and kept careful family records there is every reason to believe the pedigree is a highly accurate representation of the occurrence of the traits in the family.

Hyperkeratosis of the nails is a defect that exhibits a number of clinically distinct forms. Usually it involves keratinization of both finger and toe nails and may or may not be accompanied by shedding. In a large collection of cases by Cockayne,[4] included under the title, "Hypotrichosis et Dystrophia Ungium," the nails of fingers and toes are very thick, longitudinally striated and discoloured while the hair on the body is usually very fine and slow growing. Presumably no shedding occurs. According to Clousten[3] there are more than 6,000 Canadians with this defect, probably largely descendants of a French family that came to Canada about 180 years ago. Cockayne's collected data show that this condition is inherited as a simple dominant trait. There are a number of closely related forms of hyperkeratosis of the nails in which the finger and toe nails are extremely thick and cylindrical in form, assuming the shape of a claw and standing out at about a 45 degree angle when long. The thickened nails are brittle and dry, readily becoming infected, after which shedding usually occurs. In the family described by Walter and Bradford[9] there were 27 affected persons in five generations. In 20 of these the keratosis involved only the thumb and index finger nails. In a pedigree given by Clemente[2] an affected father transmitted the defect to five of his nine children. In this family the condition is present in all the finger and toe nails, but shedding does not occur. In a case of onychomadesis in a French family described by Montgomery,[6] the nails became yellowish in color over the lunula and gradually loosened and came out. Within from three to eight months new nails, normal in appearance, grew in. These in turn were shed within a few months. The condition was present in a woman, her son and her two brothers. Other pedigrees of hyperkeratosis, onychomadesis and onychogryphosis of the nails have been published.[1,4,5] A study of these leads to the conclusion that probably large numbers of genes are concerned in the development of the nails, and that mutation in these may result in a variety of nail abnormalities, often associated with other ectodermal defects.

The condition described here differs from all of the above and probably represents a genetically distinct abnormality. In all of the affected persons the hyperkeratosis involved only the nails of the larger toes. This may be seen in Figure 6. The condition usually makes its appearance shortly after puberty, prior to which time the nails appear normal and no shedding occurs. With the onset of the trait, the large toe-nails become from two to five millimeters thick and can be cut only with great difficulty. One of the affected persons uses bone scissors, although soaking in warm water is the most frequent method used to facilitate cutting. There is no keratinization of the nail bed. The thickened nails are yellowish in color, so that the lunula does not show, and have a tendency to become

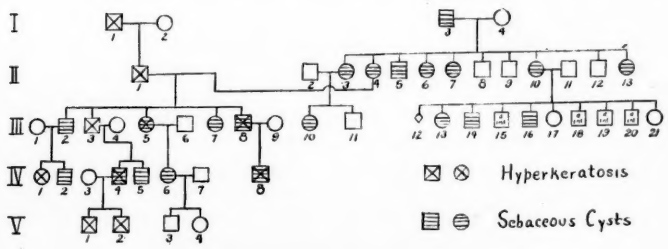


BIG TOENAILS AFFECTED

Figure 6

Individuals in this family affected with hyperkeratosis have thick bone-like nails of the big toes, which are shed periodically. The other toenails are normal and the trait varies in time of onset and severity of expression.

infected. In most of the cases the nails of the large toes are shed from time to time, new nails making their appearance soon afterward. These become just as thick as the old ones and in turn are also shed. The length of the cycle of shedding and regeneration varies from individual to individual; thus III-8 shed his nails about four times yearly, whereas his son (IV-8) sheds only about once a year. The onset of the condition in the father was in the sixteenth year of life and in the son in the eleventh year. On the other hand, IV-4 did not manifest the thick nails until after he was twenty and he never shed them. The trait thus appears to be variable both in its time of onset and in its expression. In one instance it was transmitted by an apparently normal individual (III-2). It is possible that the manifestation of the condition in this person was so slight that it escaped detection but this could not be ascertained. In none of the cases were the nails known to exhibit a cylindrical form or to assume the shape of a claw. As far as could be determined no other defect, ectodermal or otherwise, was associated with the condition. Inheritance appears to be that of an incomplete dominant, the vari-



POOLING OF GENETIC DEFECTS

Figure 7

One branch of the family transmitted a gene for defective toenails; the other a gene for sebaceous cysts. Several individuals in the fourth and fifth generations showed both defects, but there is no indication that they are linked in inheritance.

ability in the trait probably being the result of the influence of other genes and extrinsic factors.

Description of Cysts

The second condition exhibited by the pedigree is commonly known in the family as wens or sebaceous cysts. A sebaceous cyst is a retentiton cyst of a sebaceous gland. Since the description of the cysts in this family closely follows that given by Cockayne[4] for epidermoid cysts it is possible they belong in that category. Such growths are generally between a pea and a hen's egg in size and are frequently found in the scalp alone but may appear on other parts of the head and body as well. In the cases being considered here the cysts are found only in the scalp in all but one case. In this person (IV-4) there was one in the scalp and another on the lower third of the neck. In at least eight persons in the pedigree the cysts were large and severe enough to necessitate surgical removal. Individual II-4 had a multilocular cyst the size of a large hen's egg. This was removed and never occurred again. The cysts were always few in number and the time of appearance of the first one ranged from the eleventh year of life in IV-5 to the 35th year in III-5. Cockayne states that epidermoid cysts may not make their first appearance until the 40th or 50th year of life and that in one instance a cyst developed in a man 65 years old. This being the case, it is possible for an individual to transmit the gene for the condition and die before reaching the age at which the defect might have appeared in him. Siemens,[8] in a study of 109 cases of epidermoid cysts, found that in 34 there was a record of one or more relatives affected, while in 75 there were no known relatives who had the condition. Data collected from sibships belonging to families in which there is a history of the disease gives a ratio of 97 affected to 195 normal, a wide deviation from the 1:1 ratio expected for a dominant trait. In these families there are nine unaffected persons who had affected offspring. In the pedigree given here there is only a single conductor. The data for the family cases, therefore, tends to follow the pattern expected for incomplete dominant heredity. In a pedigree of what was called sebaceous tumours by Sedgwick,[7] but believed to be epidermoid cysts by Cockayne, affected females transmitted the condition to all of their daughters and none of their sons, while males never showed the condition and never transmitted it. This has been considered to be a possible case of attached-*X* chromosome inheritance in man.

It should be noted that V-1 and V-2 are still in their teens and may yet have cysts, and that V-3 and V-4 are still too young to show either condition. In contrast with most pedigrees of cysts of this sort the present one is marked

by the apparently high penetrating power of the dominant gene. This also appears to hold for hyperkeratosis of the nails. This may be shown by examining the ratios for each trait. Assuming that the matings giving rise to affected individuals are between heterozygotes and recessives, a 1:1 ratio of affected to normal is to be expected in the offspring. The actual ratio for sebaceous cysts is 20 affected to eight normal, and for hyperkeratosis nine affected to five normal. In either case it is apparent that probably few persons carry one of the genes and fail to be affected. As we have already seen there are two persons who presumably fall into this category. Here it must be emphasized that for traits like these, that show great variability in time of onset and in degree of severity, great care must be taken in assembling pedigree data and in making genetic analyses. Allowance must also be made for the fact that in all such material pedigrees with an excessive number of affected individuals are the ones which will be most likely to come to the attention of an investigator.

Linkage Improbable

Insufficient evidence is available to determine whether or not the two traits are linked. The facts, however, would seem to point against such a possibility or at least against close linkage. If we let *H* represent the gene for hyperkeratosis and *h* its normal allele; and *C c* the respective alleles for cysts and absence of cysts, then the mating between II-1 and II-4 can be represented by the linkage formula $(Hc)\ (hc) \times (hC)\ (hc)$. The four offspring, III-2 (assuming he carries the gene for hyperkeratosis), III-3 (assuming he carries the gene for cysts), III-5 and III-8 would be genetically $(Hc)\ (hc)$. All of them presumably mate with $(hc)\ (hc)$ individuals. If linkage were close such matings would rarely give the cross-over type $(HC)\ (hc)$ showing both traits. Of the six offspring resulting from these unions, one has hyperkeratosis, three have cysts and two show both traits. It therefore appears unlikely that any close linkage exists.

Literature Cited

1. Bauer-Fischer-Lenz. Human Heredity. New York: Macmillan. 1931.
2. Clemente, L. S. *Jour. Hered.* 19:529. 1938.
3. Clouston, H. R. *Canadian Med. Assoc. Jour.* 21:18. 1929.
4. Cockayne, E. A. Inherited Abnormalities of the Skin and its Appendages. London: Oxford University Press. 1933.
5. Gates, R. R. Heredity in Man. New York: Macmillan. 1931.
6. Montgomery. *Jour. of Cutan. and Genito-Urinary Dis.* 15:374. 1897.
7. Sedgwick, W. *Brit. and Foreign Med.-Chirugy. Rev.* 31:450. 1863.
8. Siemens, H. W. *Arch. f. Derm. u. Syph.* 144:175. 1923.
9 Walter, A. L., and W. L. Bradford. *Missouri State Med. Assoc. Jour.* 25:20. 1928.

✦

Effects of the War on British Fertility

The birth rate has fallen to a level which in the near future means a decline of population. The number of persons in this country between the ages of 14 and 64 is 33 million, and two thirds of them are engaged in the war effort, whether in the armed forces, civil defense or war industries. No country has ever before mobilized its man power to the extent we have. The effect of the war on the birth rate is interesting. Except as it causes a temporary loss of births by separation of man and wife and a permanent loss of potential fathers and mothers by violent deaths, it seems that the war may have a favorable effect on fertility in the long run by bringing about a change in values and removal of some of the deterrents to childbearing. There has been a remarkable increase in marriages. This is in part due to the large number of girls reaching the ages of 19 and 20 owing to the high birth rate which followed the last war. But more important are the allowances provided for the wives of the fighting men. The birth rate for the first quarter of 1942 was higher than for any other first quarter since 1931, in spite of the disruption of family life produced by the war, including the bombing of our cities and their evacuation on a large scale.—London Letter, *J. A. M. A.*, July 25, 1942.

AN ABERRENT RATIO IN PEROMYSCUS

R. R. Huestis and Victor Piestrak
*University of Oregon**

CLASSICAL genetics pictures phenotypic ratios as being fundamentally of two kinds: ratios to be expected when the involved genes are on different chromosomes and those to be expected when the involved genes are linked and the ratio consequently modified. In routine search for linked genes, one therefore scrutinizes F_2 and test cross progenies for significant deviations from the phenotypic ratio to be expected from independent assortment. We have been doing this with progeny ratios designed to test the interaction of a number of single-gene autosomal recessives and have found that while significant deviations from ratios to be expected from simple assortment are common, linkage, so far, appears to be a minor factor in their creation. The commonest factor causing deviations from expected ratios appears to be difference in viability; wild type mice are typically more viable than mutant types and these latter often differ from one another. Another important, although less common factor, is the tendency for some mutant types to be quite variable and to overlap the wild type in appearance. The proportion of individuals erroneously put in a wild type class because of this factor may be constant over a long period of time or may be diminished by the selection of parents possessing the mutant character to a pronounced degree or parents throwing a higher percentage of easily identified young. One linkage group has been reported in Peromyscus involving albino and pink eye (then called pallid). This was found by Sumner[7] when only three mutant types were known, a long shot in a species with twenty-four pairs of chromosomes.[2] Sumner's work was confirmed by Clark.[1]

In this paper we are reporting upon F_2 and test cross progenies in which all the factors cited above as modifying phenotypic ratios appear to be operating at once. These F_2 progenies were obtained by mating silver with flexed tail mice and mating the non-silver non-flexed F_1 young *inter se*. The test cross progeny are all the offspring of one double recessive male, obtained in the F_2 generation, mated with a number of the same F_1 females all in the repulsion phase.

Characters Used

The characters silver (*sisi*) and flexed tail (*ff*) were described by Huestis and Barto.[5] In silver the black pigment of the proximal portions of all hairs is reduced in amount and the black pigment present may be assembled in widely spaced, variable sized masses instead of the evenly spaced, uniform clumps of dark granules to be seen in normal hairs. Light silvers can be identified at a glance and nearly all silvers by an examination of the proximal portions of the hairs under low power magnification. An occasional mouse from silver parents is indistinguishable from wild type. Flexed tail mice have short tails usually with one or more permanent flexes produced by disalignment of certain caudal vertebrae. As a routine the tails of mice in all broods that may contain flexed tail individuals are felt for slight flexes which do not show plainly. In spite of any test so far employed, flexed tail parents continue to throw some young with tails that cannot with certainty be distinguished from those of wild type animals.

The young mice here tabulated were identified by the authors and to a minor degree by two student assistants, one a senior and the other a graduate student

*This research was made possible by grants from the General Research Council, Oregon State System of Higher Education.

in biology. We have compared the percentage of silver and flexed tail mice identified in similar hybrid progenies by all four observers and found it to be reasonably similar. The differences found were too small to be responsible for any of the conclusions later to be made and may very well be due to factors other than identification differences. The data have been gathered for a period of five years and the figures from the individual years are quite uniform in the largest class, non-silver non-flexed, and vary up and down in the smaller classes. With the idea of testing what silver mice we might be erroneously tabulating as wild type, about 300 young were reared from brown parents heterozygous for silver and flexed tail, silver brown mice being rather easy to separate visually from brown. The difference between the percentage of silvers produced by brown as compared with non-brown parents was 0.85 ± 1.88. We do not believe therefore that many silver mice were not identified as such. Silver mice appear to be somewhat less viable than wild type. In an experiment involving 523 wild type, brown, and silver mice with 50 per cent of the latter expected, 240 silver mice or 46 per cent was obtained.

In other experiments we have found some variability in the percentage of flexed tail mice identified as such. In an F_2 population of 287 wild type, brown and flexed mice, 69 (24.0 per cent) were flexed. In an F_2 population of 356 wild type, ivory and flexed mice, 54 (16.6 per cent) were flexed. Twenty-five per cent was the expectation in each case. In the present F_2 generation, 16.8 per cent of flexed tail mice were identified. We have supposed that both identification errors and relatively poor viability accounted for discrepancies between the number of flexed mice observed and the number expected in these various experiments. The reason for the variability in the proportion of flexed individuals in different F_2 generations is not known. The difference between the first two percentages is 3.5 times its probable error.

The F_2 generation involving wild type, silver and flexed tail mice is numerically presented in Table I. It represents the progeny of 168 F_1 wild type parents; 125 females and 43 males.

The deviations from an expected 9 : 3 : 3 : 1 ratio are large and the chi square value statistically significant,[4] *P* being less than .001. The most noticeable deviation is in the double recessive class, where one mouse was observed when 67 were expected. This mouse, a male, was later tested with flexed tail females and produced 19 flexed tail young. He also is responsible for the entire test cross generation itemized in Table II. Here again significant deviations from expectation are to be seen and a large chi square value, *P* being less than .001.

Three of the silver flexed tail females from the test cross generation have recently produced broods; one of five, one of three and one of two silver flexed young, all sired by the same male. Two young mice have died, two individuals in one brood and one in another brood have an aberrant gait; they weave or waltz when stimulated to move and are not of model size for age. This abnormality had not been previously noticed in the stock and may have no bearing upon the present problem.

Interpretations

When a considerable number of F_2 young had been gathered, it was assumed that the absence of the double

TABLE I. F_2 Generation (si f/si f) x (si f/si f).

Class	Obs. no.	Exp. no.	Dev.	Dev.^{2} / Exp. no.
Not silver or flex.	669	600.75	+68.25	7.75
Silver	220	200.25	+19.75	1.95
Flexed tail	178	200.25	-22.25	2.47
Silver flexed	1	66.75	-65.75	64.76
Total	1068	1068.00	0	76.93 = chi square $P \leq .001$

TABLE II. Test cross (si f/si f) x (si c/si f).

Class	Obs. no.	Exp. no.	Dev.	Dev.^{2} / Exp. no.
Not silver or flex.	17	26.75	- 9.75	3.55
Silver	51	26.75	+24.25	21.98
Flexed tail	32	26.75	+ 5.25	1.03
Silver flexed	7	26.75	-19.75	14.58
Total	107	107.00	0	41.14 = chi square $P \leq .001$

recessive class was due to a lethal effect of the double recessive combination for the ratio being obtained was reasonably close to 6: 3: 3: 0. The more recently gathered material makes untenable the theory of a double recessive lethal effect although it still seems probable silver flexed mice have very low viability. The test cross gives considerable indication that silver and flexed tail are linked, for here the presumptive non-crossover silver and flexed tail classes make up 78 per cent of the test cross progeny. The groups are not very well balanced but this may be due to relatively poor viability in the flexed classes together with some normal overlap with the present methods of identifying flexed tail mice. The matter can probably be settled by obtaining a test cross progeny from females in the coupling phase but it will take some time to gather the necessary data from these. None has bred yet.

Summary

1. A F_2 generation of over 1,000 deer mice produced by parents carrying the autosomal recessive genes silver and flexed tail in the repulsion phase consisted of about 63 per cent wild type, 21 per cent silver, 17 per cent flexed tail and 1 silver flexed tail individual.

2. A test cross generation of 107 mice consisted of 78 per cent silver and flexed tail mice and 22 per cent non-silver, non-flexed tail and silver flexed tail mice; the presumptive non-crossover and cross-over classes respectively if linkage exists.

3. Both these ratios deviate significantly from the ratios to be expected upon a basis of independent assortment of the genes for silver and flexed tail.

4. It is tentatively concluded that these ratios are the result of differential viability, some overlapping of the flexed tail class with the wild type and linkage between silver and flexed tail.

Literature Cited

1. Clark, Frank H. *Contrib. Lab. Vert. Genetics,* Univ. of Mich., No. 7, 1-11. 1936.
2. Cross, J. C. *Cytologia* 8:408-419. 1938.
3. Dice, L. R. *Amer. Nat.* 67: 1-4. 1935.
4. Fisher, R. A. and F. Yates. Statistical Tables for Biological Agricultural and Medical Research. 90 pp. Oliver and Boyd, London. 1938.
5. Huestis, R. R. and Elizabeth Barto. *Jour. of Heredity* 25:219-223. 1934.
6. ————. *Jour. of Heredity* 28: 73-75. 1936.
7. Sumner, F. B. *Amer. Nat.* 56:412-417. 1922.

◆

THE NEED FOR "GENE CONSERVATORIES"

Hans Gruneberg

Biometry Department, University College, London; at Mount Vernon Hospital, Northwood, Middlesex

THE activity of geneticists has led to the discovery of numerous gene differences. When you visit a laboratory, you are shown the genes that have been discovered and worked with in that place, and their number is often very impressive. Instructive though this may be for a visitor, there is a different side to this picture. The accumulated material clutters up the laboratory, and increasing space, assistance and money is needed each year to preserve the heritage of the past. As departments and their budgets do not grow correspondingly, this happens at the expense of research activities; so gradually that nobody notices research laboratories are turned into museums and research workers into curators. I have seen this happening on a small scale within the short period of nine years in my own laboratory, and on a much larger scale in

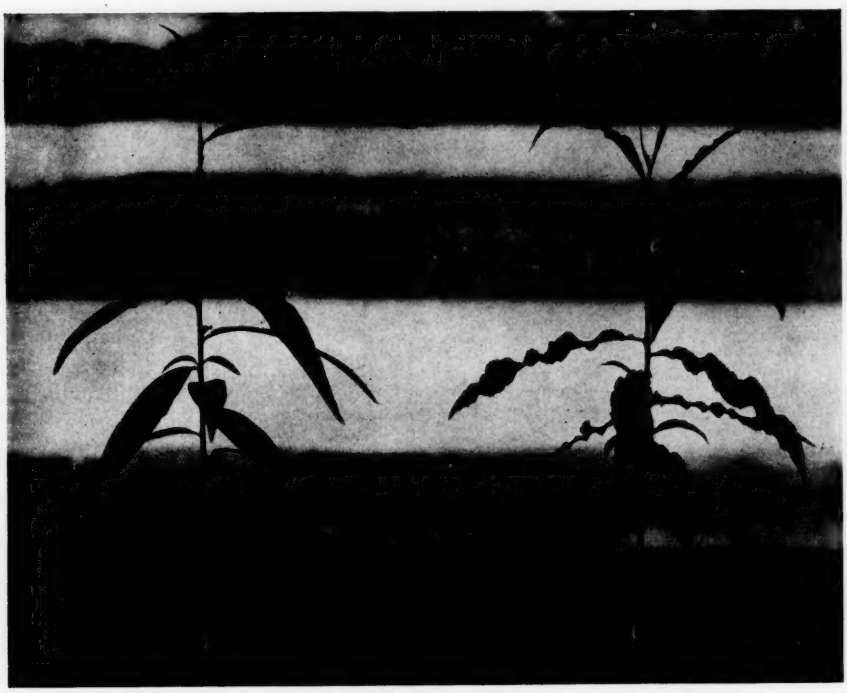
various institutions with which I am familiar. No doubt the same is true everywhere. We are walking down a one-way street, metabolizing without excreting, so to say. A new research worker may be an asset to his department when he arrives, but his legacy will assuredly be a liability to the laboratory when he leaves. Genetics is now just over 40 years old, and most departments of genetics are younger. One shudders to think what they will look like in the year 2000 A.D. Unless something is done about it, genetic research will be increasingly hampered by the products of past generations. This note, it is hoped, may induce colleagues from other laboratories to suggest solutions, and a workable scheme may ultimately emerge from a discussion.

What has happened in the past? Some departments have, or course, patiently borne the burden. Others have tried to get rid of material, with which nobody was working any more, and which seemed either unimportant or unpromising. I myself am guilty of such a case, and I shall probably never cease to regret it. Professor Egon Pearson, in 1924, published a short note (*Nature*, vol. 114, p. 433) which drew attention to the occasional occurrence of cataractous animals in a certain mouse strain; a selected strain was eventually turned over to our laboratory in 1933. As the condition manifested itself late in life and very irregularly, and as the animals were poor breeders and fairly large numbers were required to keep the stock going, it was eventually discarded. Meanwhile, and unknown to me at that time, Cohrs, (*Arch. fuer Augenheilk.*, vol. 107, pp. 489-550, 1933) described a retinal anomaly in the mouse sometimes associated with cataract (stock now extinct). Later still, a retinal degeneration with cataract was discovered in the rat (this JOURNAL, vol. 30, pp 130-1939). We shall never know whether Pearson's mice were genetically the same as Cohrs' strain, nor shall we know whether in this instance the homologue of the rat gene was lost for good. Pearson's account would have been of value, had the work with the strain been continued; with the stock lost it is utterly useless. Thus the discarding of the stock not only resulted in the loss of irreplaceable material, but also in the killing of a paper. Such "corpses" are unfortunately numerous in the genetical literature. Whenever a gene is lost, a thread is cut which can never be taken up again. If nothing has been published about it, the loss is small. But if we put something on record, we thereby become responsible to those who read our account. We are bound to misjudge the importance of our material very often. In the case of Pearson's mice, the remorse followed the deed very soon; often it will be many years before later work is handicapped because a certain animal or plant mutant cannot be revived.

We are on the horns of a dilemma. We must keep all our mutants going; but if we do, our laboratories get filled with junk, and research is hampered. Hence we must create an outlet, a kind of depository for our mutants. No single laboratory, no single university can undertake the job. Perhaps no single country can. It is clearly a matter which calls for centralized planning. At present, universities, learned societies, and research foundations are in fact unknowingly supporting a very inefficient scheme for the maintenance of our mutants as carried out by individual departments. The scheme is inefficient, because some mutants are kept with unnecessary replications, whilst there is no guarantee against the loss of others. Centralization is admittedly difficult, but it is certainly not impossible. Universities or research departments might be quite willing to subscribe to a scheme which would enable them to "farm out" some of their material. The State and private institutions annually spend large sums of money on the preservation of wildlife. I am the last to question the desirability of such undertakings; but I venture to suggest that the benefit to the public, by way of furthering agricultural and medical research, of maintaining our mutants, would be at least as great as keeping the bison alive.

THE INHERITANCE OF WAVY-LEAF CHARACTER IN THE PEACH

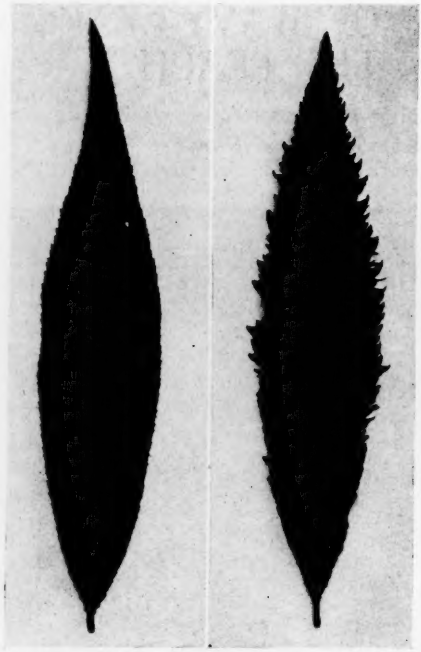
D. H. Scott and F. P. Cullinan
Division of Fruit and Vegetable Crops and Diseases,
U. S. Department of Agriculture, Beltsville, Maryland



SEEDLINGS OF GOLD DROP PEACH SELF-POLLINATED
Figure 8

Plant on left has normal leaf type; plant on right has wavy leaves. The wavy type is recessive.

THE normal leaves of most peach trees are relatively smooth with little crinkling, and have crenate or separate margins (Figure 9), except for varieties such as Early Crawford, which have leaves that are sinuate and puckered along the midrib. During the course of the peach breeding work carried on at this station it was noticed that in the 1938 selfed progeny of the Gold Drop variety there were a few seedlings having very wavy leaves (Figure 8), deeply serrated. Their foliar glands were seemingly globose. These leaves were unlike the normal leaves of peach varieties. In the 1939 F_2 progeny of Carman $\times$ Gold Drop, obtained by selfing a selection from the F_1 generation, seedlings with this same wavy-leaf character were observed. Most of the seedlings possessing the new character are definitely undesirable because they tend to



NORMAL AND WAVY LEAVES

Figure 9

Peach leaves from seedlings of Gold Drop self-pollinated. The leaf on the left is the smooth, normal type; that on the right is the wavy type with deeply and irregularly serrate margin.

be weak growers, are of dwarf habit, and set fruit poorly.

The wavy-leaf seedlings from the 1938 Gold Drop selfed progeny flowered fully for the first time in 1941. Pollen was collected from one seedling and used to make the Gold Drop × wavy-leaf seedling and (Carman × Gold Drop) × wavy-leaf seedling crosses indicated in Table I. The reciprocal crosses were not made because the trees with wavy leaves have never matured fruit. One wavy seedling which was selfed set one fruit, but it dropped off before it was ripe.

The seeds resulting from these crosses were germinated in the greenhouse and the seedlings were potted individually into 4-inch pots and grown in the greenhouse four months before setting in the field.

Results

Table I shows the genetic behavior of the wavy-leaf character in the various progenies. The ratios, except that for the

Table I. Inheritance of wavy-leaf character.

Parentage	Seedlings with		χ^2	Theoretical monogenic ratio	Number seeds	Percent germination
	Smooth leaves	Wavy leaves				
Gold Drop x Carman, F_1	83	0	-	-	132	62.9
Carman x Gold Drop, F_2	135	41	0.27	3:1	221	79.6
(Carman x Gold Drop) x wavy-leaf seedling	122	173	6.25	1:1	412	86.1
Gold Drop selfed	34	11	0.01	3:1	58	77.6
Gold Drop x wavy-leaf seedling	185	186	11.19*	1:1	492	63.2

*χ^2 for 1 degree of freedom = 6.635 P .1%

TABLE II. Vigor as indicated by height of peach seedling progenies containing plants with wavy-leaf character. Seedlings measured 30 days after being potted.

Parentage	Leaf character	Height of seedlings in centimeters															Number of seedlings	Average height
		2-3.9	4-5.9	6-7.9	8-9.9	10-11.9	12-13.9	14-15.9	16-17.9	18-19.9	20-21.9	22-23.9	24-25.9	26-27.9	28-29.9	30-31.9		
Gold Drop x wavy-leaf seedling	Smooth										5	7	4	3	1		20	23.35 ± 0.53*
Do.....	Wavy	1	6	8	5	8	13	5	4								50	10.22 ± 0.52
(Carman x Gold Drop) x wavy-leaf seedling	Smooth		4	3	11	18	45	50	32	12	3						179	13.64 ± 0.24
Do.....	Wavy	13	32	62	50	12	1										170	6.79 ± 0.14
Carman x Gold Drop, F_2	Smooth		4	3	19	23	25	25	14	11	9	2					135	13.14 ± 0.34
Do.....	Wavy	5	19	12	3	1											40	5.32 ± 0.27
Gold Drop x Carman, F_1	Smooth				1		4	3	7	8	4	8	7	1	3	1	47	19.97 ± 0.72

*Standard error

Gold Drop × wavy-leaf seedling cross, fit a simple monogenic inheritance, with the smooth-leaf character dominant to the wavy condition, and with Gold Drop and the F_1 selection from Carman × Gold Drop each heterozygous for this leaf character.

The progeny of Gold Drop × wavy-leaf seedling does not give the anticipated 1:1 segregation, but it should be noted that the germination of the seeds in this group is considerably less than in most of the other progenies. The ratio departs from expected in having too few wavy-type seedlings in relation to smooth-leaf type. The wavy-leaf seedlings are much less vigorous in growth than the smooth-leaf seedlings, as is indicated in Table II and it is assumed that many of the weakest wavy seedlings failed to grow, thus causing the deviation from the expected 1:1 ratio. In each of the other crosses where wavy-leaf seedlings appeared in the progeny, there is also a shortage of plants having this character, and, although the discrepancies are only slight in the progeny, the trend is similar to that found in the Gold Drop × wavy-leaf seedling cross that had low seed germination.

Connors[2] has previously shown that the reniform glands of the peach are dominant and glandlessness is recessive; globose glands represent a heterozygous condition in a blended type of inheritance. He states that a mixed gland condition (two different types) on the same tree has never been found by him. In the peach breeding work at Beltsville, over 5,000 seedlings having all types of glands have been examined and the mixed gland condition has never been observed. Reniform glands on weakly vegetative plants appear globose, but when the plant is stimulated into vigorous growth, the glands are reniform. Bailey and French[1] reported as follows:

> The Gold Drop variety has reniform foliar glands, which is supposed to be a homozygous gland condition Among 954 seedlings of Gold Drop selfed there appeared 213 or 22.3 per cent having globose glands and very sharply serrated, wavy leaves, a character usually associated with eglandular leaves. At present no data are available to explain this behavior.

The leaf type observed by Bailey and French is apparently the same as that observed by the authors. It appears, however, that in the development of the wavy-leaf type the shape of the reniform gland is modified to make it appear globose. The glands on the margin of the wavy leaves are generally stalked and appear to be globose, but when sessile glands are found on the pedicles of the same leaves they are reniform. The deeply serrate margins of the wavy leaves do not appear to be the same as the serrations usually associated with eglandular leaves. The margins of the wavy leaves are irregularly serrate as compared to the evenly serrate margins on eglandular leaves.

Summary

The wavy condition of the leaf is controlled by a single pair of alleles with the wavy factor recessive to the smooth-leaf condition.

The wavy-leaf character is undesirable and can be detected in the seed flat and eliminated from field plantings.

Literature Cited

1. Bailey, J. S. and A. P. French. *Ann. Report. Mass. Agr. Exp. Sta., Bul.* 305. 1934.
2. Connors, C. H. *Proc. Amer. Soc. Hort. Sci.* 18:20-26. 1921.

A BOOK OF THE PIGEON

A Review

DOMESTICATION of the pigeon extends back into remotest antiquity, when pigeon-keeping seems to have been associated largely with religious rites. Doubtless pigeons were kept also for other interests, for the wild species early became differentiated into numerous types resembling more or less closely our present-day breeds and varieties. Certainly the pigeon "fancy" was a well developed art in many parts of the world long before there was any adequate literature on the subject. The year 1735 stands as an important milestone in columbophilism, for in that year was published the first real pigeon book, the *Columbarium: or the Pigeon House. Being an Introduction to a Natural History of Tame Pigeons,* by John Moore. Pigeons must have been a hobby or avocation with Moore for he made his living as an apothecary and was widely known in London in his time as "The Worm Doctor of Abchurch Lane," being mentioned by Pope, Swift and other writers of the day. However that may be, he obviously knew his pigeons, and produced a work which was widely copied, paraphrased and plagiarized by later writers. To pigeon men Moore's book was sufficient to immortalize him in spirit, even though Pope was less optimistic about his mortal body, as is indicated by his lines:

> O learned Friend of *Abchurch-Lane,*
> Who sett'st our entrails free!
> Vain is thy Art, thy Powder vain,
> Since Worms shall eat ev'n thee.

During the century and a half following the *Columbarium* a good many works on pigeons, good, poor and indifferent, appeared in England, among which some of the best known are *A Treatise on Domestic Pigeons,* usually referred to simply as the *Treatise,* published anonymously in 1765, with numerous and varied later editions; *The New and Complete Pigeon Fancyer,* by Girton (1780-90?); *Doveçote and Aviary,* Dixon (1851); *A Treatise on the Art of Breeding and Managing Tame, Domesticated, Foreign, and Fancy Pigeons,* Eaton (1858); *Pigeons: Their Structure, Varieties, Habits, and Management,* Tegetmeier (1868); *The Illustrated Book of Pigeons, with Standards for Judging,* Fulton (Edited by Wright, 1874-); *Fancy Pigeons,* Lyell (1887). *Fulton's Book of Pigeons,* an enlarged edition put out by Wright in 1895, has long held the field as the standard work on varieties, their breeding and management. There have been many less comprehensive books, treating of single breeds or subjects, both in England and in this country, and from the standpoint of detailed experiment and consideration of origin, there is, of course, Darwin's classical work reported in *The Variation of Animals and Plants under Domestication,* first published in 1868. A book, *Nűzliches und vollständiges Taubenbuch,* published anonymously at Ulm in 1790 was somewhat comparable in Germany to the *Columbarium* in England. Since that time, and particularly during the nineteenth century, there have appeared a number of comprehensive and extensively illustrated works in a number of countries and in different languages. Curiously, however, few if any were forthcoming in the fifty years following Fulton's *Book of Pigeons.* The most recent work of note in this country up to last year was *The Book of the Pigeon,* by Naether (1939), which can be commended for originality, readableness, and general accuracy.

Just a trifle over two hundred years after Moore's *Columbarium,* comes another book, which sets a new high and which is destined long to be a landmark in pigeon literature. This is *The Pigeon** by Wendell M. Levi, a lawyer

*LEVI, WENDELL MITCHELL. The Pigeon. xxxii + 512 pp. 785 illustrations. With preface by Dr. Oscar Riddle. $10.00. Printed by the R. L. Bryan Company, Columbia, S. C. 1941. Published by the author, to whom orders should be sent.

in Sumter, South Carolina, who has had a lifelong and varied experience with pigeons. The title page refers to his service as a Captain in the Pigeon Service of the U. S. A. Signal Corps in France during World War I, his editorship of the Questions and Answers department of the *American Pigeon Journal,* his presidency of the National Pigeon Association and of the Palmetto Pigeon Plant. It does not mention that boyhood love of pigeons which was the start of it all and which has made him a pigeon enthusiast ever since. That in itself is not so unusual, for most enthusiasts and fanciers started in the same way; what is unusual is the catholicity of his interests, which is reflected in his book. Previous pigeon books have stressed particularly the fancy breeds and have given lengthy discussion on their characteristics and how to breed for them, this being based on individual or collective experience but with little or no understanding of the underlying principles. Levi does not treat so exhaustively of the various breeds, although all of those known in this country and some others are discussed, and nearly all of them are illustrated by excellent photographs of actual specimens rather than, as is usually the case, by drawings of ideals which are never attained. The discussions do not attempt to give complete descriptions and breed "standards," but they do present a general picture of the breed, with pertinent facts as to origin, relationships and peculiarities. For greater detail the breeder or fancier of any particular variety of pigeon will doubtless want to supplement what he gets here by some work dealing specifically with the breed of his choice.

The Pigeon differs from previous books in that the latter mostly considered pigeon keeping only as an art and gave scant attention to scientific aspects of the subject; in this, however, their authors can hardly be held to blame for there was little pertinent science to be reported at the time they were writing. In their discussion of breeding, for example, they did not have the advantage of the knowledge of pigeon genetics which has been accumulated in the last forty years. This is not to say that the present knowledge of pigeon genetics is adequate to enable a breeder to produce directly and infallibly a prize-winning bird with all the many fancy points of color, feather, shape and carriage that are demanded; but it is sufficient to provide him many short-cuts and to help him to know in what he can proceed directly and in what else he must still depend to a considerable extent on good judgment combined with good luck. It is sufficient also to show the ineffectiveness of the lengthy discussions on how to breed this or that character, based on individual experiences, which constituted so much of earlier works and still occupies much space in the pigeon journals. Readers of the *Journal of Heredity* will accordingly be particularly interested in Chapter V, which deals with "Genetics — Variation and Heredity," and which gives an excellent account and summary of the work that has been done on the genetics of the pigeon up to the present time. As in other chapters, Levi has shown a breadth of knowledge of published work and contact with specialists in the various subjects that is surprising in one who makes no claims himself to being a scientist. In the chapter on Genetics he has been fortunate in having the able assistance of W. F. Hollander, who has himself made significant contributions in that field. Again the subject is well illustrated with photographs as well as a colored plate to show some twenty-eight color combinations. This should be helpful but unfortunately even yet by the use of color photography and reproduction exact values are seldom obtained and hence the pictures cannot be taken too "literally."

The chapter on Genetics is followed by one on Practical Breeding which contains suggestions that should be helpful. The geneticist may not be quite satisfied with the definition of line breeding given, but that is of small moment in the present connection.

Space will not permit of more than mention of many other outstanding features of the book. The treatment of

Anatomy is more or less standard, but collects a good deal that has not previously been brought together in one place. That on Physiology, like the one on Genetics, is a penetrating review and shows the effects of close contact with Oscar Riddle and his outstanding work in this province. Under Behavior is discussed the reproductive cycle and other habits. The discussion of homing summarizes interesting facts but leaves the problem still unsolved. The use of pigeons as messengers in war is given considerable attention, including stories and photographs of birds which distinguished themselves on the battlefields of France. The chapters on Diseases and on Feeds and Feeding report the latest findings in these subjects and are supplemented by the author's extensive experience. They should prove almost indispensable to the pigeon breeder, who will find occasion to refer to them constantly. The author's personal experience comes in strongly also in the remaining chapters on Housing, Commercial Squab Production and The Fancy Pigeon—Exhibiting, Racing, Training, Organizations, etc. Under the last head are sections dealing with the training of pigeons for such activities as racing, night-flying, high-flying and tumbling, treated again with the hand of a master.

Much thought has obviously been given to the arrangement of the book. The sections, each consisting of one to several paragraphs, are numbered consecutively and are extensively cross-referenced, which assists the reader in getting an overall view of a particular subject. No classification of the multifarious breeds is attempted, and perhaps wisely, for the author recognizes that any classification must be largely artificial and often inconsistent. He has accordingly avoided that difficulty by arranging them alphabetically, which is generally satisfactory but has some incongruities. As previously mentioned, the book is profusely illustrated throughout and with pictures which have a definite purpose and pertinency. It is remarkably well documented, the bibliography occupying 14 pages and comprising probably 900 or more references. And there is an adequate index. One other feature which is a token of the care that has been expended on the work is that it is remarkably free of typographical errors. All in all, it is obviously a "labor of love," and incidentally it is a book anyone interested in pigeons or in subjects related to those treated cannot afford not to have readily at hand. It is necessarily somewhat expensive but is well worth the cost. One wonders, indeed, that it can have been produced at the price.

LEON J. COLE

University of Wisconsin

ON THE SEX RATIO IN CATTLE*

JOHN W. GOWEN
Iowa State College

IN 1917 the writer[2] presented data on the sex ratio of 2,023 cattle births. These observations have been increased to 3,559 births. As these data were taken in a manner to insure a completely random sample without selection, it seems desirable to publish the full record.

Materials and Methods

The data for this investigation were the product of the work of Dr. Raymond Pearl and the writer while at the Maine Agricultural Experiment Station. The following arrangements were made to insure a group of completely random sample cattle births. Something over 200 dairy cattle farmers were invited to cooperate in the investigation by preparing and sending to the experiment station a sheet showing the date, name of the bull and cow and other pertinent information for each service performed in their dairy herds.[5] This sheet was filed at the Maine Agricultural Experiment Station immediately after the service took place. The cooperator, when the birth appeared, filled in a similar sheet showing the date and sex of the birth as well as the name of the sire and dam, etc. The service and birth sheets were brought together at the Maine Station, each service sheet being traced to show either a resulting birth, an unsuccessful service or that the cow was slaughtered before the birth of the fetus.

The Secondary Sex Ratio

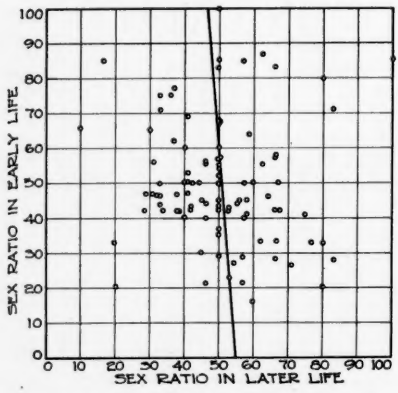
The tabulation of these records shows that in 3,559 births there were 1,799 males and 1,760 females. This ratio indicates a nearly equal proportion of males and females in cattle births. The divergence from equality comes in a slight excess of male births, the per cent of males being 50.5 and of females 49.5.

In 1932 Johansson[3] published an extended study of sex ratios in cattle in which he summarizes the results of previous workers as well as furnishing extensive new data of his own. These new records were taken from the records of individual farmers. The data were thought to be a true random sample of the sex ratio for the particular breed studied, even though they lack some rather essential controls. The sex distribution for three Swedish and three Finnish breeds was 63,960 males to 60,206 females or a percentage of males of 51.5. Data of other investigators, cited by Johansson, showed sex ratios in individual herds, ranging from 48.4% males to 51.9%. Whether this range represents true random variation of the sex ratio or as Johansson points out, an extension of the range toward lesser males by the quite customary habit of omitting to record females is, of course, an unanswered question. The data of Jewell on the intra-uterine sex ratio of 1,000 cattle at different stages of development show an average sex ratio of 55.2% males. The larger sex ratio was found for the youngest and for the oldest foetuses.

Genetic Elements in Sex Control

The equality of the sexes throughout these data suggests that there are no major genetic elements other than the familiar chromosome mechanism which are causing deviation in the sex ratio, unless perchance these deviations are balanced in either end of the sex distribution. With such a balancing certain parents would have a preponderance of male offspring and other parents would

*Journal Paper No. J-1036 of the Iowa Agricultural Experiment Station, Ames, Iowa. Contribution from the Iowa Agricultural Experiment Station and the Maine Agricultural Experiment Station.



SEX-DISTRIBUTION WITHIN SIRES

Figure 10

Relation between the sex ratios of the first half of a sire's progeny with that of the last half, sires with eight or more in their families.

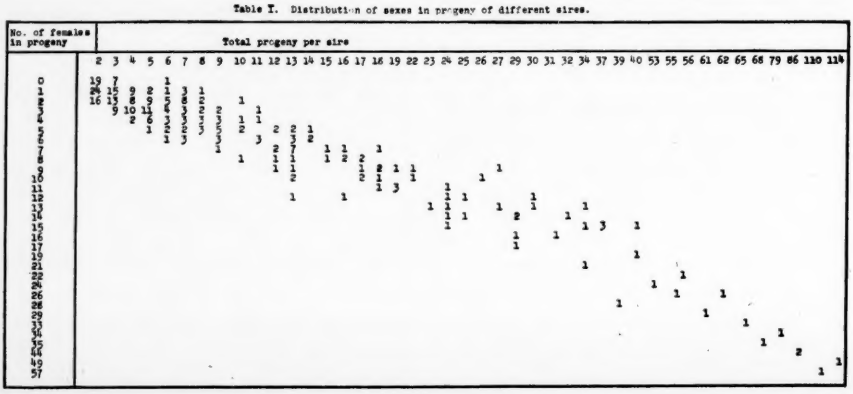
show a corresponding preponderance of female offspring. These extreme types should be more frequent than would be expected by chance. The work of King[4] shows that a genetic basis for such a divergence of the sexes within strains does exist for certain species with an *XY-XX* chromosome mechanism of sex determination. By selections for a high preponderance of males in one line and a corresponding high frequency of females in another, King was able to split a group of rats into two lines differing in their sex ratio. The first six generations of the experiment were without selection. No significant changes in the sex ratios of the two lines were noticed during this period. In the next four generations selection was practiced and the sex ratios diverged from the previous median value of roughly 51% males. From the tenth generation on there was little change, the sex ratio of each line remaining at the level established and characteristic of that line. The percentage of males for the high male line for the eighth to the twenty-fifth generation was 55.1%. That for the line where the females were selected for was 44.9. Interestingly enough, this major change in the sex ratios of the two lines was accomplished in not over two generations of selection and when once established remained essentially constant.

Drosophila offers other cases of genetic control of the sex ratio[1,6,7] through control of the maturation behavior of the *X* and *Y* chromosomes. With special

TABLE II. Distribution of Sexes in Progeny of Different Dams.

No. of ♀'s in progeny	Total progeny per dam										
	2	3	4	5	6	7	8	9	10	11	12
0	125	30	3	1		1					
1	257	112	13	5	1						
2	123	84	27	5	5	1	1		1		
3		28	16	4	3		2	1	1		
4			1	3	2	1	2	1		1	
5				1		2		2	2	1	
6							1		2	1	
7							1				
8											1
9									1		

Table I. Distribution of sexes in progeny of different sires.



inheritance the sperms formed largely contain *X* chromosomes thus leading to a large proportion of females in the progeny.

There are cattle breeders who feel that sires differ in their ability to produce one sex or the other in their progeny, in a manner comparable to the differences which have been demonstrated for rats or Drosophila. In their opinion, certain bulls are female-producing whereas other bulls tend to have male progeny. Differences of this kind should become apparent if we tabulate the frequency of the sexes for given sized families and test these frequencies against their proper chance distributions. These distributions have been formed for both sires and dams which have two or more progeny.

The data in Table I and in Table II show no marked departures from the binomial distribution of the sexes in the different sized families. This would agree with the assumption that the sex of the progeny is determined through a series of events which distribute themselves at random. This conclusion leads to the view that within these data there are no sires which tend to produce either male or female progeny in excess. These conclusions would apply particularly to the individual having families of less than eight individuals since beyond this number the data become too few for adequate testing. The data of Johansson also bear out this observation.

Beyond families containing eight individuals it is possible to rearrange the material further to test this random distribution of the sexes within the family, as well as to bring out another point. If it be admitted that there are genetic influences which affect the sex ratios in cattle then it would follow that these influences should play their part throughout the entire progeny-producing period. It is possible, therefore, to test this hypothesis by dividing the progeny of sires which had more than eight individuals in each of their families into two parts—one half of which were born in the younger period of the sire's life and the other half when the sire was older. By comparing these two parts — one with the other as shown in Figure 10, it is possible to determine whether or not any consistent influence exists throughout the sire's life tending toward the production of one sex or the other. If such an influence exists there should be a correlation in the sex ratio during the two periods.

The distribution of the sexes for the sires with different sized families is essentially random when we compare the sex ratios of the first half with that of the second half of their lives. The correlation coefficient for this comparison is —0.09. It is true that the dot diagram shows a few stragglers in the group which have a high frequency of females in both periods of the sires' lives. It is also true that a few sires have a high male frequency in both the earlier and later parts of their families. However, an examination of the table shows that there are approximately equal numbers in the corresponding group where the sex ratio changed sides in the second half of the sires' life, high sex ratios during the first part of their lives and low in the second or low in the first part and high in the second part of their lives.

Summary

The data from these observations thus seem to indicate that the distribution of sexes in cattle families is essentially at random in most instances. The arrangement of the data in Figure 10 is such as to lead to the conclusion that the age of the sire or dam does not materially affect the sex of its progeny.

Literature Cited

1. Gershenson, S. *Genetics* 13:488-507. 1928.
2. Gowen, John W. *Ann. Report of the Maine Agricultural Expt. Station.* pp. 205-228. 1918.
3. Johansson, I. *Zeitschr. f. Tierzuchtung and Zuchtungsbiologie* 24:183-268. 1932.
4. King, H. D. *Jl. Expt. Zool.* 27:1-37. 1918.
5. Pearl, R. *Ann. Report of the Maine Agricultural Expt. Station.* 1914.
6. Sturtevant, A. H., and Th. Dobzhansky. *Genetics* 21:473-490. 1936.
7. Sturtevant, A. H. *Proc. Nat. Acad. Sci.* 23:360-362. 1937.

THE SIZE AND SHAPE OF HUMAN HEAD HAIR

Along Its Shaft

HENRI C. SEIBERT AND MORRIS STEGGERDA
Carnegie Institution of Washington
Cold Spring Harbor, Long Island, N. Y.

IN the literature on cross sections of human head hair, there often are allusions to the fact that the hair varies in size and shape along its shaft, but actual data as to the manner in which it varies are surprisingly rare. During a study to survey racial differences in hair cross-section undertaken in this laboratory we were careful to make the sections at a definite distance from the scalp in order that our results be comparable.[5] To establish the necessity of such a procedure, long hair samples were selected for sectioning and the results are presented in this paper.

Six samples of hair were taken from American Dutch white females living in Holland, Michigan, and nine from Maya girls of Yucatan, Mexico. The first group consists of elderly women from 50 to 70 years of age, while the Indian girls were between 10 and 20, with the exception of one who was 45. The sixteenth sample was from the head of a white female of 35 years and of American parentage.

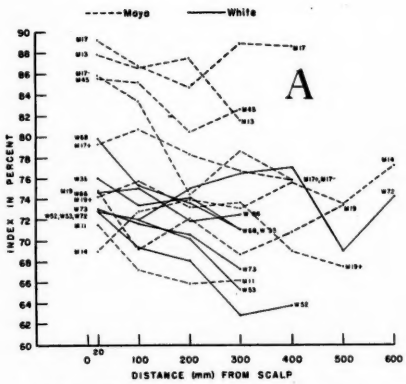
The technique described by Steggerda[4] and Steggerda and Seibert[5] was followed throughout. One exception was made to the usual procedure in that all the hairs in the sample were selected to reach a minimum length and those hairs falling short were discarded. Consequently from 80 to 100 hairs, averaging 400 to 500 millimeters in length, were laid out, fastened together firmly, and then sectioned at 20, 100, 200, 300, 400 millimeters from the original cut. If the sample was long enough, cuts were made at 500 and 600 millimeters. The sections were mounted, drawn, and measured in the usual manner carried out in this laboratory.

Kneberg[2] cut one wavy hair, 30 centimeters long, and another straight hair, 19 centimeters long, at intervals of one centimeter along the entire shaft. She found that the index for the former varied from 75 to 100 and for the latter, 63 to 90. Unfortunately it was not stated in what manner the variation occurred. However, in another paper[3] she related that hair sections varied in size according to where the shaft was sectioned. In that study one straight hair was cut at three centimeter intervals, the sections drawn with a camera lucida, and their area measured with a planimeter. The areas in square micra were as follows: from base to tip: 4382, 4350, 4720, 4510, 4302, 4734, 4286, 4334, 4955, 5051, showing a remarkable variation in size of one shaft at different levels.

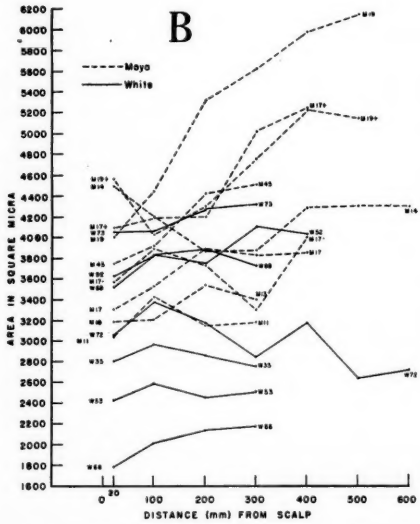
Results

An inspection of Table I and Figure 11 *A* of our data shows that the area at 100 millimeters is more than at 20 millimeters for all samples but two, namely, M19+ and M14. This is true irrespective of age or of race of the individuals. At the next one hundred millimeters the trend is not so obvious; seven of the samples show a decerease, and in the successive intervals, the trend is even more irregular. Some samples show remarkable increase in size as M19 and M17+, others show irregularity as M14, M17—, and W63. These changes are summarized in Table II.

Not all the samples fit precisely into this pattern, but it is evident that the majority of cases are concentrated in the groups I, II and III, showing the hairs have a tendency to increase rather than decrease in area.



Index of long hair samples at increasing distances from the scalp.



MAYA AND WHITE HAIR INDEXES

Figure 11

The upper chart shows the hair index and the lower chart the hair area for six samples of white hair and nine samples of Maya Indian hair. The hairs change in size and shape over their entire length.

It is interesting to note that Kneberg's samples (loc. cit.), if her sections at 30, 90, 120, and 300 mm. are considered, would fall in class III. In all but three cases (M14, W72, W35) the hairs are larger in area at their terminal cut than at the basal region.

The maximum diameter follows very closely the pattern of the area and for that reason is omitted from graphic form.

The index, however, shows a tendency to decrease as the hair progresses from the scalp. From Figure 11 *B* and Table I, it is seen that all but four of the hair samples decrease in index between 20 and 100 mm., but after 100 millimeters the trend is not consistentl as only ten samples show decreases between 100 and 200 mm. These trends are summarized in Table II.

TABLE I. Size and Shape of Hair along its Shaft.

	Area*							Index						
	20	100	200	300	400	500	600	20	100	200	300	400	500	600
W73	40.4	40.6	42.6	43.2				73.0	71.7	70.6	67.3			
W72	30.6	33.9	31.8	28.8	31.8	26.4	27.2	72.8	72.0	75.0	76.4	77.0	69.0	74.2
W68	35.2	38.2	38.9	37.3				79.9	75.3	73.6	71.0			
W66	17.9	20.1	21.3	21.8				74.7	75.1	71.9	72.5			
W53	24.3	25.9	24.6	25.1				72.9	72.0	70.2	65.3			
W52	36.2	38.3	37.5	41.0	40.3			72.8	69.4	68.1	62.8	63.8		
W35	28.0	29.7	29.6	27.6				76.0	73.4	76.1	71.0			
M45	37.5	39.2	44.2	46.1				85.6	85.2	80.4	82.6			
M19	40.0	44.5	53.1	56.2	59.7	61.4		74.8	69.3	72.1	68.7	70.8	73.5	
M19+	45.6	40.2	43.0	47.6	52.1	51.4		74.2	78.7	72.2	73.6	69.0	67.5	
M17	33.1	35.3	36.9	38.3	38.6			89.2	86.8	84.7	86.8	88.5		
M17+	40.9	41.9	42.0	50.1	52.3			79.3	80.7	78.3	76.7	75.8		
M17-	35.7	38.9	37.3	33.0	40.0			85.9	83.4	76.4	78.6	75.8		
M14	44.9	42.0	36.7	38.7	42.9	43.1	43.1	69.0	72.8	73.9	73.0	75.8	73.4	77.2
M13	31.9	32.1	35.4	34.0				87.9	86.6	87.6	81.5			
M11	30.5	34.3	31.5	31.8				71.5	67.2	65.9	66.1			

*Square micra divided by 100

TABLE II. Trends in the area and index of hair cut at various levels along the shaft.

Area

I	M17+, W73, M19, M45, W66
II	W68, M17, M13, M11, W35
III	W52, M17—, W72, W53
IV	M14, M19+
V	
VI	

I—Steady increase
II—Increase then decrease
III—Increase then decrease followed by increase
IV—Decrease then increase
V—Decrease then increase followed by decrease
VI—Steady decrease

Index

M17+, M19+
W66, M14
M17, M45, W52
M13, M17—, W35, M19, W53, W72
W68, W73, M11

Again the samples do not fit exactly into the six groupings, but the result indicates an opposite trend rather than that observed for the area, in that the majority of samples are now in the categories IV, V, and VI. In all but two cases the hair at the terminal section has a lower index than it had at the beginning.

Discussion

It is obvious that hair does change along its shaft both in size and shape. Kneberg's comment that individual hairs are very variable is certainly borne out by this study. The changes in size and shape can be great or small but, we believe, not entirely in a random direction. Hair is known to be elastic and hygroscopic[1] and therefore environmental factors could and do exert some influence on its shape and size. It is possible that the hair changes its shape because of combing, brushing, the use of headgear, or its own weight as it lies flat on the head. It is doubtful, however, that dressing the hair alone caused the observed changes as the Maya girls rarely do anything to their hair outside of washing it, and with the exception of W35, all the White women were past the age of artificial hair dressing and are definitely known not to have done more than wash and brush the hair.

The increase in area and the decrease in index are puzzling and mere hypothecation with no direct evidence is of little value. With a decrease in the index, one would expect a decrease in area as the circle contains the greatest area for a given perimeter, but since the reverse is apparently true, one must conclude that in spite of its wide fluctuations, the hair actually expands in size as it progresses from the scalp.

Literature Cited

1. Danforth, C. H. Hair. Amer. Med. Assn., Chicago. 1925.
2. Kneberg, Madeline. *Amer. Jour. Phys. Anthrop.* 20:51-68. 1935.
3. ———. *Amer. Jour. Phys. Anthrop.* 21:279-286. 1936.
4. Steggerda, M. *Jour. Heredity* 31:474-476. 1940.
5. Steggerda, M. and H. C. Seibert. Size and shape of head hair from six racial groups. *Jour. Heredity* 32:315-318. 1941.

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks.

Its membership is composed of men of science, teachers, publicists, physicians, clergymen, parents, students, horticulturists, and breeders of live stock throughout the world.

The Association owns the JOURNAL OF HEREDITY, which is published monthly and sent to each member without additional cost. Every member is thus a part owner of the JOURNAL.

Membership imposes no burdensome obligations. The Association, which is co-operative in nature, welcomes assistance in research, but does not demand it. Members are invited to submit discussions of the results of their research, accompanied by new and unusual photographs. All papers received will be given full consideration by the editorial board.

Manuscripts should be sent to the Editorial Office of the JOURNAL, Victor Building, 724 Ninth St., N. W., Washington, D. C.

Proof: In order to facilitate prompt publication only galley proof will be sent to authors.

Reprints: On request the author will receive gratis 100 reprints without covers. Covers and additional copies will be furnished at cost.

REQUIREMENTS FOR MEMBERSHIP

Subject to the approval of the Council, any person interested in the improvment of the human race or the creation of better varieties of plants and animals, is eligible for membership.

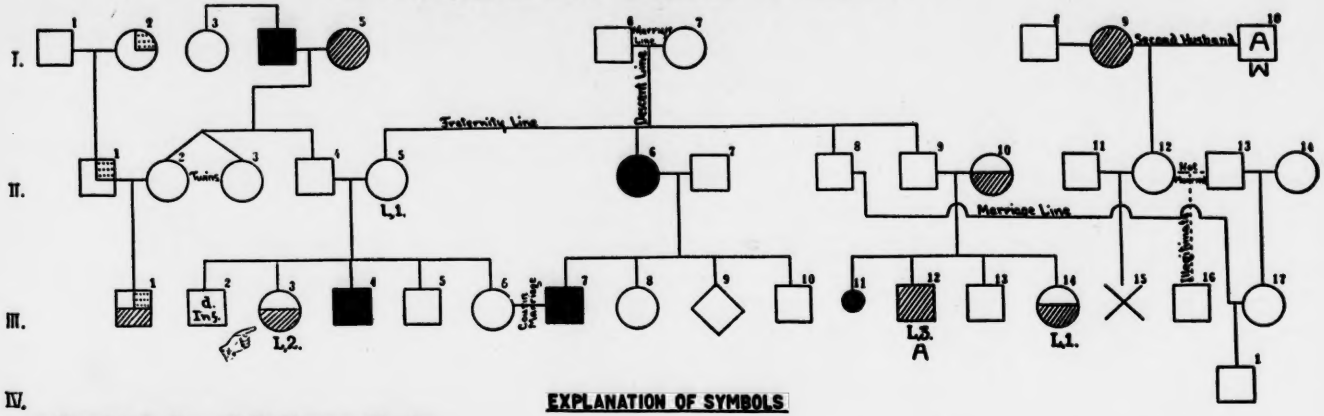
The Association welcomes all who are interested in its program, and the Secretary will be glad to answer any inquiries.

Annual dues, giving the right to attend all meetings, and receive the JOURNAL OF HEREDITY, are $3 within the United States and its possessions; $3.25 in Canada, and $3.50 in all other foreign countries; life membership, $50. Subscription to the JOURNAL is $3.50 per year (foreign postage extra).

If you are not already a member, and want to become one, or if you know of anyone who would be interested in membership, write to

THE AMERICAN GENETIC ASSOCIATION
Washington, D. C.

SAMPLE PEDIGREE CHART SHOWING THE MANNER OF CONSTRUCTION, AND THE USE OF STANDARD AND SPECIAL SYMBOLS.



EXPLANATION OF SYMBOLS

(a) **Standard for all Pedigree Charts:**

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d. Inf.=Died in Infancy; Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III. 7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic, F, feebleminded; I, insane, M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic, Sx, sexually immoral; S, syphilitic, T, tuberculous; W, wanderer.

(b) **Especially devised or selected for this particular pedigree-chart:**

■ ● = Successful leaders in politics. ▣ ◔ = Extra thumb on right hand. L.1. = Highly successful author. L.3. = Little or no ability in literary efforts. ▨ ◍ = Superior in vocal music. ▨ ◍ = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

Prepared by the International Commission of Eugenics. See *Journal of Heredity* for August 1926. Additional copies may be obtained from A.G.A.